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ABSTRACT

This document contains the reports of research at a symposium on "Altered States of Consciousness and Alcohol." The participants primarily agreed that alcohol induces an altered state of consciousness similar to other drugs, but that this phenomenon has not been explicitly stated due to the current interest in newer and more novel drugs. The presentations included in this report are: (1) Objective and Subjective Effects of Alcohol on the Ascending and Descending Limbs of the Blood Alcohol Curve; (2) Alcohol and Secobarbital: Altered States of Consciousness Assessed by an Information Processing Approach; (3) Brain Damage in Alcoholics: Altered States of Consciousness; (4) Physiologic Concomitants of the Alcohol State: Arousal or Relaxation; (5) Biological Rhythms and Alcohol Effects: Altered Rhythms of Consciousness; and (6) Voluntary Control of Blood Alcohol Levels: An Attempt to Manipulate States of Consciousness. The report concludes with a general discussion on alcohol and its effects on the body. (Author/PC)

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ALTERED STATES OF CONSCIOUSNESS AND ALCOHOL

A SYMPOSIUM

Presented at the Southwestern Psychological Association (SWPA) Meetings

May 3, 1974

El Paso, Texas

By the Alcohol Research Staff

Oklahoma Center for Alcohol-Related Studies

Oklahoma City, Oklahoma

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I. ALTERED STATES OF CONSCIOUSNESS AND ALCOHOL

Ben Morgan Jones, Ph.D.
University of Oklahoma Health Sciences Center

INTRODUCTION

The title of this symposium is "Altered States of Consciousness and Alcohol." The participants are primarily investigators who have wroked together for the past several years at the Center for Alcohol Related-Studies in Oklahoma City. We feel that alcohol induces an altered state of consciousness similar to other drugs, but that this phenomenon has not been explicitly stated due to the current interest in newer and more novel drugs. We hope this symposium will be valuable to both researchers and clinicians in interpreting the acute and chronic effects of alcohol in terms of how and why a person manipulates his state of consciousness with alcohol. This symposium will allow us to present some of our new research and reinterpret some of our past research. We hope it will be informative and stimulating to those individuals who wish to reevaluate the commonly held interpretations concerning the effects of alcohol on behavior.

When we refer to an altered state of consciousness, I think we have in mind a definition similar to that proposed by Arnold Ludwig in Charles Tart's book entitled, "Altered States of Consciousness." Dr. Ludwig defines altered states of consciousness as "any mental state, induced by various physiological, psychological or pharmacological maneuvers or agents, which can be recognized subjectively by the individual himself (or an objective observer of the individual) as representing a sufficient deviation in subjective experience or psychological functioning from certain general norms for that individual during alert, waking consciousness. This sufficient deviation may be represented by a greater preoccupation than usual with

internal sensations or mental processes, changes in the formal characteristics of thought and impairment of reality testing to various degrees" (Tart, 1969).

Andrew Weil, in his book, The Natural Mind, discusses what he calls the fact that "we seem to be born with a drive to experience espsodes of altered consciousness." Ue uses as examples of these small children who whirl themselves around and around and fall to the ground in order to experience an altered state (Weil, 1973).

Regardless of the exact definition or nature of altered states of consciousness, it is the purpose of this symposium to discuss the possibility of alcohol producing some type of altered state. If individuals drink alcohol to produce some type of altered state, then this may give us a new insight into the reinforcing properties of alcohol that may lead to alcohol addiction and chronic alcoholism.

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II. OBJECTIVE AND SUBJECTIVE EFFECTS OF ALCOHOL ON THE ASCENDING AND DESCENDING LIMBS OF THE BLOOD ALCOHOL CURVE

Ben Morgan Jones, Ph.D.
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Alcohol is classified, pharmacologically, as a depressant drug. However, the observed behavior immediately following a given dose of alcohol can be classified as stimulant-like behavior. As we all have observed, most social drinkers become more talkative after drinking and quite frequently act elated and happy, often laughing and giggling at what appears to be inappropriate times. However, after one stops drinking, his blood alcohol level begins to decline and much different behaviors are observed. Now the euphoric feeling starts disappearing, the individual states he no longer feels intoxicated, and becomes rather tired and depressed. Data from our laboratory, as well as reports by Ekman, Frankenhaeuser, Goldberg, Bjevier, Jarpe, and Myrsten, 1963, in Stockholm, Sweden, indicate the happy, euphoric and intoxicating behaviors occur while the blood alcohol is rising (ascending limb). After the peak blood alcohol level is reached and the blood alcohol level begins to decline, (descending limb) then the intoxicating effects of alcohol disappear rapidly and are replaced by feelings of sleepiness, tiredness and overall behavioral depression. Thus, we have termed the period of the rising blood alcohol level which is associated with stimulant-like behavior such as talkativeness, euphoria and intoxication as the ascending limb. The period of a decreasing blood alcohol level that produces a depression of behavior such as tiredness and sleepiness, we have called the descending limb. As you might guess, most individuals at parties consume an alcoholic beverage rapidly to get the intoxicating feeling and then continue to sip to maintain a slowly rising blood alcohol level. Once a person stops drinking and starts to decline in blood alcohol level, he feels he is sober long before his blood alcohol level is down to zero. This is a

dangerous time to try to drive since the subjective feeling of intoxication does not correspond to ability to carry out driving skills.

Until recently, most alcohol studies have not taken into consideration possible performance differences on the ascending and descending limbs of the blood alcohol curve, although earlier studies had reported such differences do exist (Goldberg, 1943; Eggleton, 1941). Recent evidence indicates that male social drinkers perform more poorly on the ascending limb than on the descending limb at comparable blood alcohol levels for reaction time (Young, 1971), abstract reasoning (Jones and Vega, 1972) and verbal memory (Jones, 1973).

With this background, I am going to show you a video tape of a "Social Party" we had at the Oklahoma Center for Alcohol-Related Studies to give you a better idea of the behavioral effects that are different on the ascending and descending limbs. As you view this tape, you might ask yourself several questions: 1) Does alcohol produce an altered state of consciousness? 2) Is this state of consciousness different on the ascending and descending limbs of the blood alcohol curve? and 3) How are these altered states similar and different from other drug states?

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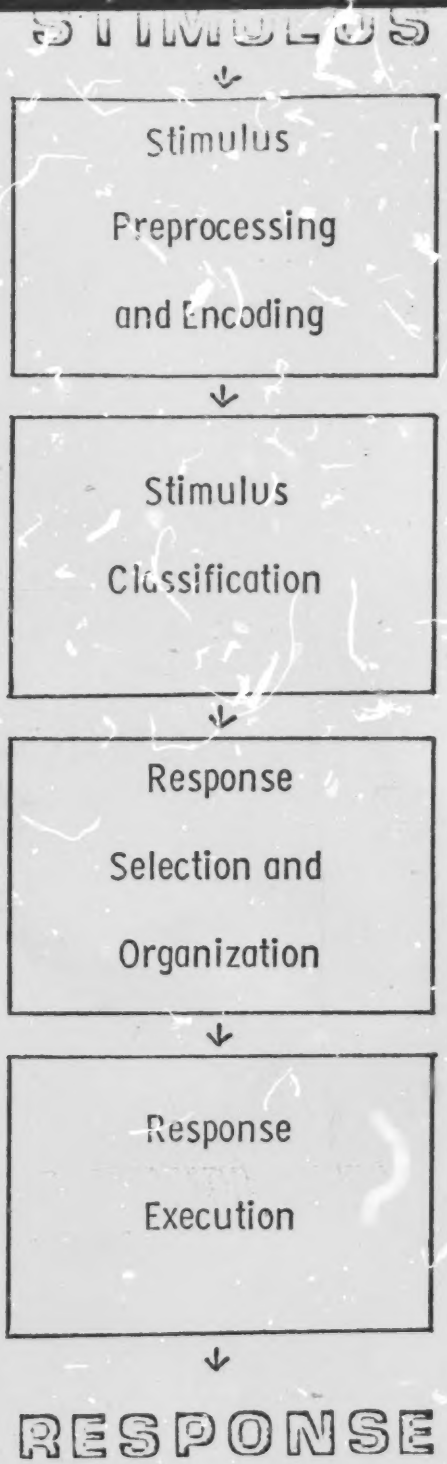
III. ALCOHOL AND SECOBARBITAL: ALTERED STATES OF CONSCIOUSNESS ASSESSED BY AN INFORMATION PROCESSING APPROACH

Van K. Tharp, Jr. and O. H. Rundell, Jr.
University of Oklahoma Health Sciences Center

In this presentation I am going to first introduce an information processing model and show how it can be used to interpret altered states of consciousness. Secondly, a method for determining how various drugs affect the operations in the model will be explained, and results using two drugs in three experiments will be briefly summarized. These results will then be interpreted with respect to altered states of consciousness.

A new approach to assessing the effect of various psychoactive drugs is to consider the action of the drug with regard to a generally accepted model of human information processing. The first slide (Figure 1) illustrates the most widely accepted model (Smith, 1968) and the one used in our analysis. In simple tasks in which a response is required to each stimulus, the model postulates four essential operations or stages. These include, in sequence: (1) stimulus preprocessing at a sensory perceptual level in which a stimulus is interpreted and encoded in a neural form; (2) stimulus-categorization, wherein the neural representation is compared with items stored in memory; (3) response selection and organization in which an appropriate response is chosen; and (4) response execution.

The use of an information processing analysis enables us to define an altered state of consciousness (or ASC) in a very specific manner. Instead of a shift in subjective experiences, an ASC becomes a change in information processing. Thus, one drug might impair a particular stage in the sequence and produce a distinct ASC. A second drug might impair a different operation,

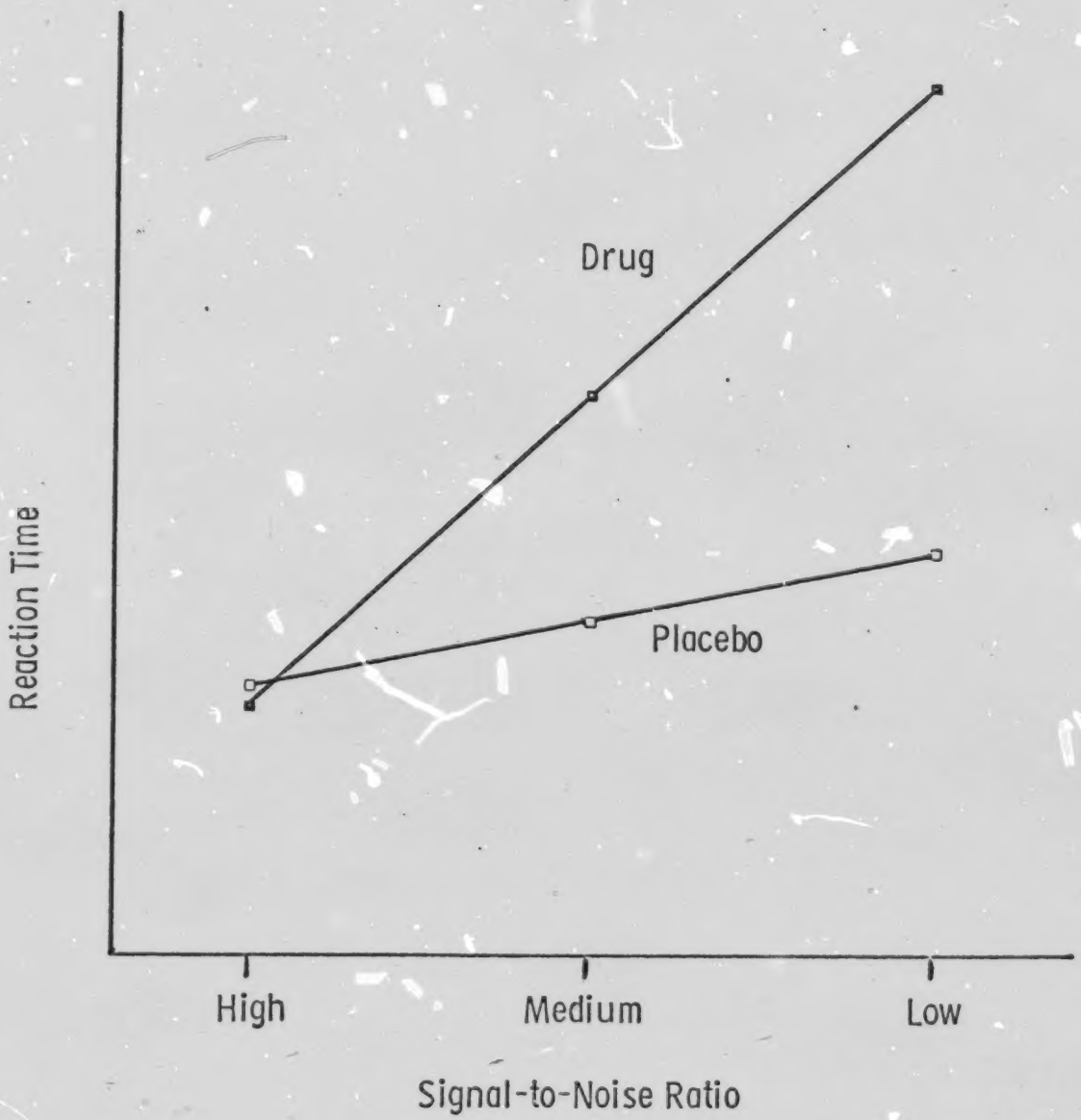


The four primary operations in sequence which are postulated by Smith (1968) as being necessary to process information.

FIG. 1

producing a different ASC. A third drug might impair two stages, while a fourth drug might completely change the sequence of operations in the model. Again, both drugs would produce different ASCs. This conceptualization of an ASC, while interesting, would be quite useless without a means of determining how various psychoactive drugs (or other psychoactive variables) act upon different stages in the above model. Fortunately, Sternberg (1969) has provided such a means.

Basically, a drug is introduced into a multiple treatment design in which the experimental variables have each been established by prior research to affect different operations in the model. These variables will be called <u>established treatments</u> in the remainder of this paper. One can then examine how a drug affects each established treatment in order to determine whether or not the drug impairs the operation in the model associated with that particular treatment. According to the Sternberg analysis, if a drug has its greatest effect at the most difficult level of an established treatment and its smallest effect at the easiest level, then it probably is affecting the same stage as that treatment. Let us look at an example. (Figure 2) represents a hypothetical experimental paradigm in which we are measuring the time required to repeat letters played by a tape recorder at a constant level of loudness. These letters, however, are played against a background of white noise which varies in loudness, thus varying the relative signal-to-noise ratio of the stimuli. Signal-to-noise ratio is an established treatment which has been shown to affect the initial stage in the model (Biederman & Kaplan, 1970; Sternberg, 1967) -- stimulus preprocessing. Note in the example that the drug has no effect at the highest signal-to-noise ratio (i.e., the least impaired signal) and its greatest effect at the lowest signal-to=noise ratio (i.e., the most impaired signal). This relationship, called a positive interaction, suggests that the drug



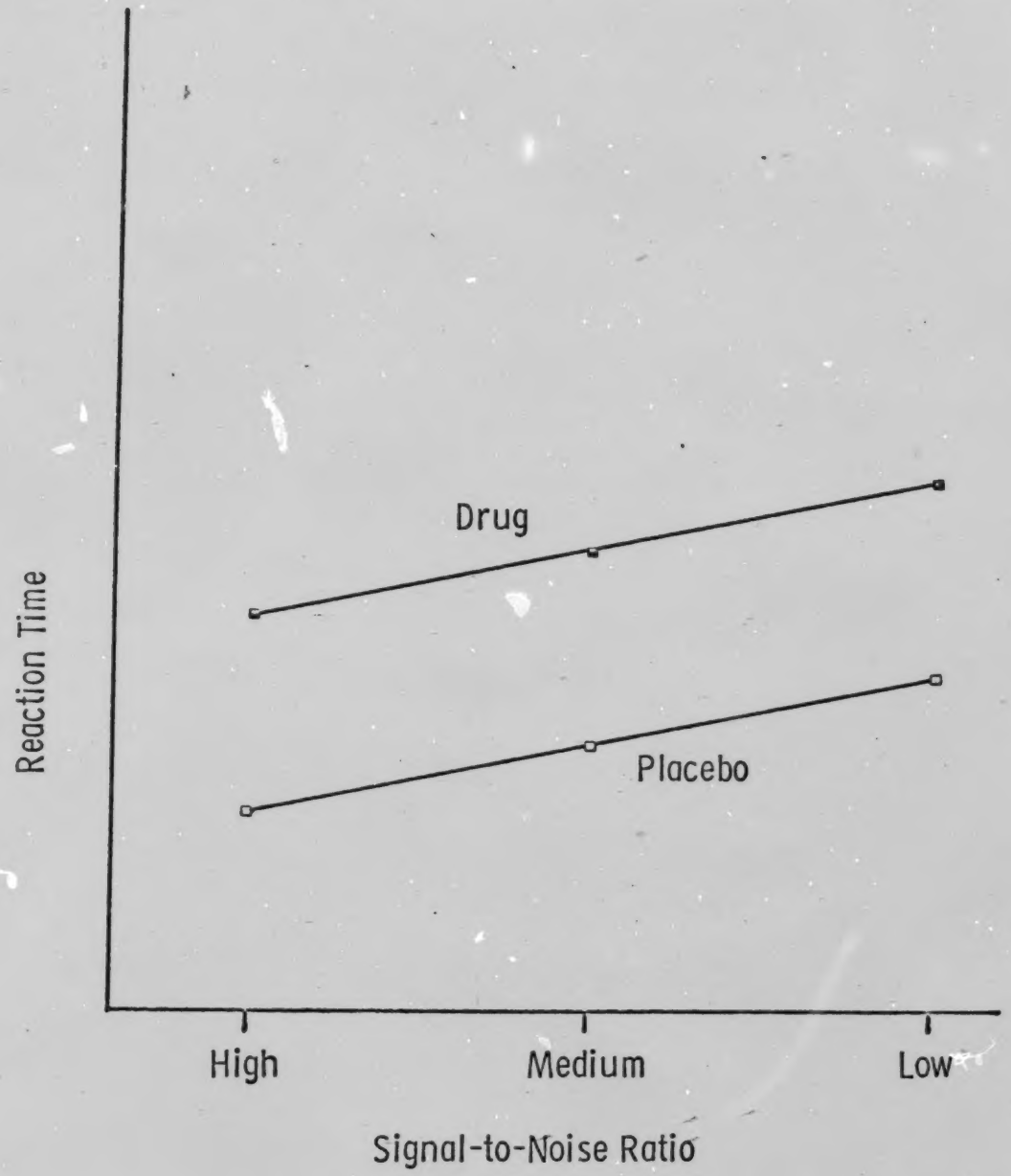
A hypothetical example of a positive interaction between the effects of a drug and an established treatment. This relationship suggests that the drug effect is mediated by the same information processing stage as the established treatment.

FIG. 2

effect is proportional to the effect of the established treatment. Thus, according to Sternberg, the drug effect is probably mediated by the same information processing stage as the established treatment. Since signal-to-noise ratio has been linked to the stimulus preprocessing operation by prior research, this operation is probably responsible for the most or all of the drug effect.

Sternberg's second assumption is that if a drug adds a constant amount of impairment to each level of an established treatment, then it is probably affecting some stage other than the one associated with that treatment. Again, let us look at an example. In this slide, (Figure 3) signal-to-noise ratio is again the established treatment. Note that the drug adds a constant increase to the reaction time at each level of signal-to-noise ratio in the placebo subjects. Thus, the drug effect seems to be realtively independent of the effect of signal-to-noise ratio. According to Sternberg, then, the stimulus preprocessing stage of the model, which mediates the effect of this established treatment, is not particularly vulnerable to the effects of this drug. The overall effect of the drug, then, is probably due to some other operation required of the task.

In summary, then, the method introduces a drug into an experimental design with several established treatments. One can then determine the locus of effect of the drug with respect to the model by examining how the drug effects these treatments which have already been linked to a particular stage of the model. A positive interaction between the effects of a drug and an established treatment suggests that the drug impairs the stage tapped by that particular treatment. In contrast, if the effects of the drug and established treatment are independent (additive), then the drug is not particularly vulnerable to the information processing stage associated with that treatment.



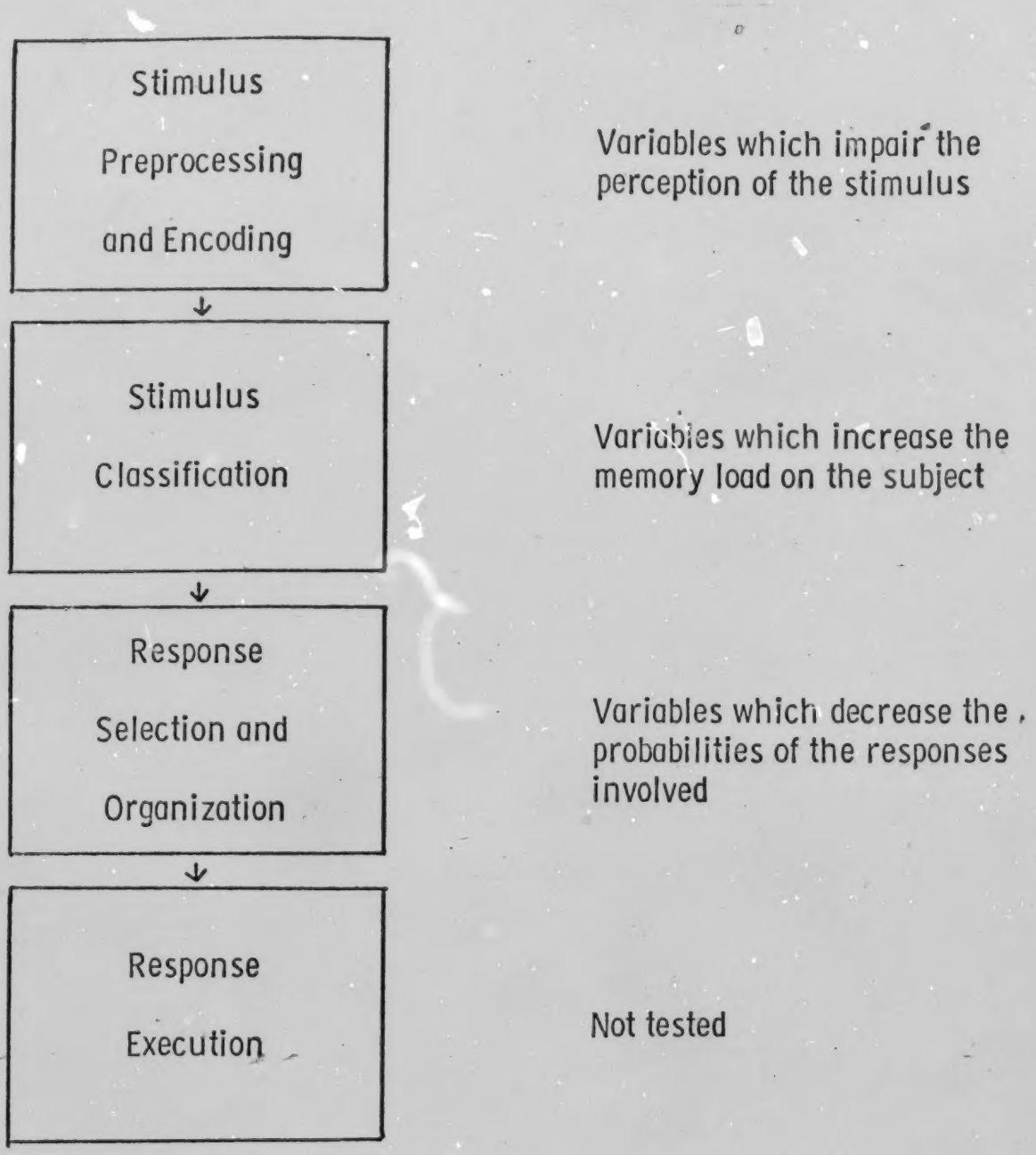
A hypothetical example of an additive relationship between the effects of a drug and an established treatment. This relationship suggests that the drug effect is mediated by a stage other than that which mediates the effect of the established treatment.

FIG. 3

Using this rationale, then, we have introduced two different drugs -- alcohol and secobarbital -- into a series of three core experiments with several established treatments. Alcohol and secobarbital were chosen because both are depressant drugs which usually produce an initial euphoria. In addition, both drugs are addictive and show a good deal of cross tolerance. Nevertheless, Overton (196) has shown that rats, at least, can discriminate between drug conditions.

The next slide (Figure 4) shows the four-stage model and the variables which affect each operation in the model. Stimulus preprocessing and encoding is tapped by variables which impair the perception of the stimulus. In our three core experiments these variables included signal-to-noise ratio (as described in the examples) and a visual equivalent of superimposing a checkerboard grid over a visual stimulus as a mask. The second stage of the model was only tapped once in our core experiments by a manipulation which varied the size of a memorized set against which a probe was compared. Finally, response-selection and organization was sampled by several manipulations of response probability. These manipulations experimentally varied the individual's expectancy for each response. We have not yet tested the response execution stage of the model.

In our core experiments, there were three possible results with each drug. First, a drug could consistently interact positively with the effects of treatments mediated by a particular information processing stage. This would suggest that the drug probably impairs that particular stage. Secondly, the drug could alter the relationship normally found between the established treatments. For example, if two variables which affect a particular information processing operation are used in the same experiment, then they should interact. Administering one of the drugs might eliminage this interaction. This pattern of results would suggest that the drug somehow


HYPOTHETICAL STAGES
ESTABLISHED TREATMENTS
Stimulus Preprocessing and Encoding
Variables which impair the perception of the stimulus
Stimulus Classification
Variables which increase the memory load on the subject
Response Selection and Organization
Variables which decrease the probabilities of the responses involved
Response Execution
Not tested

Variables (established treatments) used in our core experiments with their associated hypothetical stages.

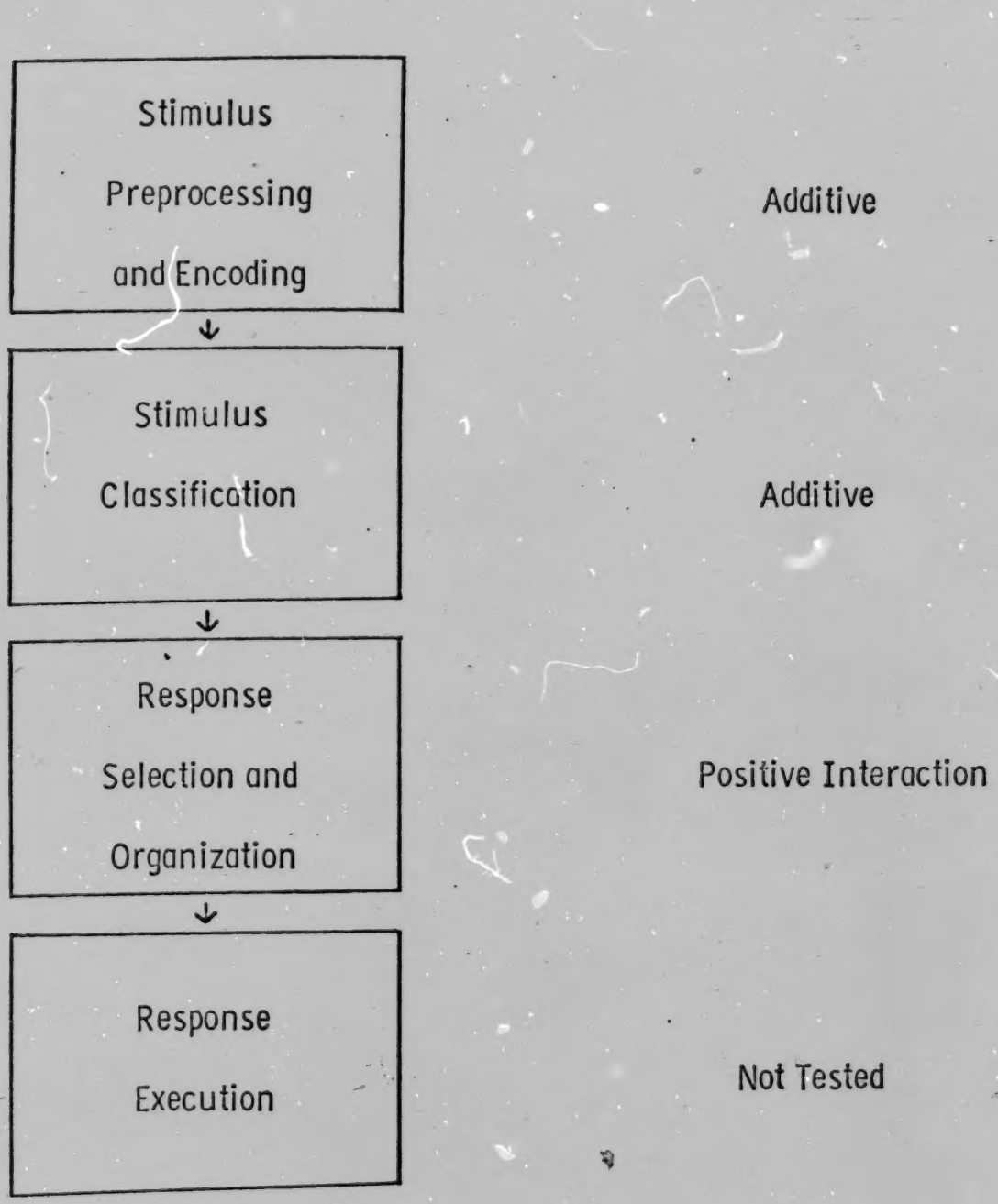
FIG. 4

rearranges the way information is processed. Finally, the psychoactive agent could show a different pattern of results in the three experiments and thus question the validity of using an information processing approach in drug research.

The next slide (Figure 5) shows the overall pattern of results when a moderate dose of alcohol (blood alcohol content - 100 mg%) was introduced as a variable in our core experiments. Basically, the effects of alcohol consistently interacted with treatments associated with the operation of organizing and selecting an appropriate response. This stage then appears to be a probably site of action of the drug. Furthermore, the alcohol effect was independent of the effects of all of the other established treatments. The other operations in the model, therefore, do not appear to be particularly vulnerable to the effects of this drug.

When secobarbital (200 mg/70 kg) was introduced into our core experiments, a different pattern of results was found. The next slide (Figure 6) shows that secobarbital interacted with the effects of variables associated with both the stimulus-preprocessing and encoding stage and with the response selection and organization stage. Therefore, both of these operations appear to be probable loci for a secobarbital effect. The bartiturate, like alcohol, did not appear to affect the stimulus classification stage.

In summary, alcohol consistently interacted in all experiments with treatments implicated in one particular stage of information processing--that is, response selection and organization. Although the alcohol dosage was large for moderate drinkers, the drug effect was independent of the effects of all other treatments. Furthermore, we observed no changes in the relationship between established treatments in any of the experiments when the drug was introduced. Thus, alcohol does not appear to change the sequence of stages. What the drug does is induce an altered state of consciousness



Summary of the effect of alcohol on established treatments in our three core experiments.

FIG. 5

HYPOTHETICAL STAGES	SECOBARBITAL EFFECT ON ASSOCIATED ESTABLISHED TREATMENT
Stimulus Preprocessing and Encoding	Positive interaction
Stimulus Classification	Additive
Response Selection and Organization	Positive Interaction
Response Execution	Not Tested

Summary of the effect of secobarbital on established treatments in our three core experiments.

FIG. 6

associated with an impaired outputting operation -- response selection and organization. Secobarbital, on the other hand, consistently interacted with treatments mediated by the stage of response selection and organization, as well as with those mediated by stimulus preprocessing. Again, we observed no changes in the relationships between variables known to affect particular stages, suggesting that secobarbital also does not alter the sequence of stages in the model. What the drug does is induce an altered state of consciousness associated with both an impaired inputting operation --stimulus preprocessing-- and with and impaired outputting operation--response selection and organization. Thus, although alcohol and secobarbital are both depressant drugs with many similarities, they probably each induce a distinct "altered state of consciousness" as defined earlier in this presentation.

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IV. Brain Damage in Alcoholics: Altered States of Unconsciousness [1]

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Abstract

Research in our laboratories and the results of many other investigators has led us to propose two hypothesis of neuroanatomical - neurophysiological disturbances in the brains of chronic alcoholics. First, many of the behavioral deficits appear to be most readily explained by disturbed frontal-limbic connections. Alcoholics appear to have deficits in maintaining "sets" leading to correct performance on non-verbal but conceptual tasks, a finding reminiscent of frontal-lobe damaged animals. Second, other neuropsychological deficits appear to be in those functions governed by the right hemisphere, i.e., tasks involving visual-spatial conceptual or perceptual-motor performance. Comparisons of groups of alcoholics on verbal tests of intelligence (Shipley-Hartford) and visual spatial tests (Raven's Progressive Matrices) indicate deficits in the latter but not the former in long term alcoholics. Both hypothesized disruptions, the right hemisphere and the frontal limbic, result in altered behavior of which the alcoholic remains largely "unaware."

The general theme of this symposium is that alcohol in acute doses leads to altered states of consciousness. In the case of chronic alcoholism, I submit that they can best be understood as altered states of unconsciousness. Before being accused of playing with words, let me remind you that consciousness remains a rather slippery and ill-defined concept (Globus, 1973). Most definitions of consciousness refer to a focus of awareness, a subjective searchlight which plays across a vast background of neural activity. As with the iceberg, which is mostly submerged, the preponderance of brain functioning never reaches the

rather narrow pathway we term consciousness. The famous English neurologist Henry Head (Head, 1963) pointed out how consciousness itself is often unware of what is missing. Consider, for example, the patient who has a visual field scotoma. Such a person adjusts his visual behavior automatically to compensate for the field cut. It is frequently a surprise to the patient when the loss is demonstrated. Or consider the person who has petit-mal epilepsy and picks up a train of conversation after a brief seizure as though there had not been an intervening lapse in consciousness. Recall also the altered behavior in frontal lobe patients where judgment and accountability for one's behavior is impaired to the observer but not from the patient's point of view.

Recently a new dimension of the conscious-unconscious states has emerged from the "split-brain" studies in humans (Sperry, 1968). If the human brain is divided into two largely separate hemispheres by cutting the connecting commissures (corpus callosum, anterior and posterior commissures), and if material is presented to the left and right hemispheres separately, Sperry and his colleagues have shown that the left hemisphere responds verbally and skillfully to various problems. When the same subject is presented with the same stimuli to the right hemisphere, the subject makes appropriate behavioral choices but cannot verbally explain his behavior. It is as though the behavior is not in the focus of consciousness or at least it cannot be communicated by the right hemisphere. These findings have led workers such as Eccles (1973) to postulate that consciousness is located in the left hemisphere. While this position clearly does not account for all of the facts, it does seem that consciousness is more commonly associated with the overt or covert activity which we call language.

certainly when we attempt to order our conscious activity subjectively or for the purpose of communication.

Most of the discussion so far can be summarized rather simply: subjective consciousness represents only a small part of the brain's activity so that vast portions of the brain's functioning can be disturbed without the subjective experience of consciousness ever being aware of the loss of function. It is our contention that chronic alcoholism results in neuropathological changes leading to such altered states of non-conscious brain activity.

What are the neuropathological effects of chronic alcoholism which could produce such effects? There is widespread agreement that chronic alcohol ingestion results in impaired functioning of the central nervous system (see extensive discussions in Wallgren & Barry, 1971; Seixas & Eggleston, 1973). While there is debate as to whether impairment of function or neuropathology can be uniquely attributed to ethanol, there is a growing body of evidence which suggests that alcohol does indeed have direct neurotoxic effects (Freund, 1973). Postmortem studies by Courville (1955) indicate pronounced cortical atrophy, especially in frontal-parietal areas, in chronic alcoholics. Pneumoencephalographic studies have demonstrated enlarged ventricles and cerebral atrophy (Brewer & Perrett, 1971; Feuerlein & Heyse, 1970; Haug, 1968; Shimojyo, Sheinberg & Reinmuth, 1967; Tumarkin, Wilson & Snyder, 1955). Psychiatric and behavioral indications of organic brain syndromes are frequently found in alcoholics as described in any text in psychiatry or neurology.

Research in our laboratories and the results of other investigators have led us to propose two neuroanatomical-neurophysiological disruptions in the

brain function of chronic alcoholics. The first hypothesis is that alcoholics have frontal-limbic neuropathology. Time does not permit detailing the neuroanatomical and neurophysiological data in support of this position but there is considerable evidence for such pathology, especially in air studies from the European laboratories. I should like to present some of our data in which the deficits obtained are in accord with the frontal-limbic neuropsychological hypothesis.

Our first study was one in which we compared performances in alcoholics, brain-damaged and control subjects on the Halstead Category Test, a measure of conceptual ability (Jones & Parsons, 1971). The results are depicted in Figure 1. Alcoholics were significantly impaired on the Category test compared to controls but not significantly different from brain damaged. Young alcoholics did not differ from young controls but did differ significantly from young brain-damaged. Older alcoholics were more impaired than older controls but not different from older brain damaged. The results from this experiment led us to embark on several experiments using another, and more psychometrically sound, test of conceptual behavior, the Wisconsin Card Sorting Test. In the next study (Tarter & Parsons, 1971) we found that alcoholics took more trials to reach criterion and made more errors than control Ss. Analysis of the error patterns suggested that the deficit lay in the alcoholic's tendency to interrupt correct sequences of choices. Also in this study we found that the longer the duration of drinking the more the impairment on the WCST.

We then devised a more definitive experiment in which a group of long term alcoholics (8 to 10 years of alcoholism) and a group of short term alco-

holics (8 or less years of chronic alcoholism) were compared to control Ss. The groups were equated for age and education. The results are presented in Figure 2. Cycles, on the abscissa refers to 6 sets of sorts with color, form, number sorting required in each set, seventeen shifts in all. In the trials to criterion it is very clear that long term alcoholics are impaired ($p < .01$) relative to short term alcoholics and controls. Indeed the latter are strikingly similar. Analysis of the error patterns indicates a possible explanation for the deficit. In the next Figure 3 we have plotted the percentage of each group making an error as a function of number of preceding correct choices. With a task criterion of ten successive correct choices before shifting reinforcement, it is clear that an error will greatly increase number of trials. The analysis suggests that error patterns for controls and short term alcoholics are similar but that the long term alcoholics interrupt successful runs of choices much more frequently than the other groups. Ratings of motivation based on behavior in the experiment indicated interest and motivation in all three groups at about the same level.

Another type of error analysis is that of distinguishing perseverative errors from other types of errors, i.e., when a shift in concept occurs does the S perseverate the old response? In Figure 4, it is apparent that the controls and short term alcoholics show an orderly progression toward fewer perseverative errors over cycles. The long term alcoholics manifest a significantly flatter slope although all start out at the same level initially.

It is clear that the long term alcoholics have two characteristics which interfere with their conceptual performance on the Wisconsin Card Sorting Test:

1) they make more perseverative errors and 2) they lose their "set" in patterns of correct responses. Both of these characteristics have been discussed in the literature as typical of frontal lobe animals (Warren & Akert, 1964; Konorski, Teuber & Zernicke, 1972).

Another characteristic of frontal lobe animals and patients are difficulties in inhibitory control (Warren & Akert 1964). To look at this possibility in more detail we used a task in which the S must turn a knob as slowly as possible through a 180° arc. Alcoholics were compared to controls in this experiment (Parsons, Tarter & Edelberg, 1972) and the results presented in Figure 5 were found. The alcoholics turned faster throughout the task than controls despite explicit instruction to turn as slowly as possible. The alcoholics were therefore relatively "impaired" on this test of control over behavior. Again the results are consistent with frontal lobe hypothesis noted earlier. Now to the extent that our frontal-limbic dysfunction hypothesis is valid it would appear that chronic alcoholics may well have behavioral deficits of which they are unaware and these deficits can lead to less effective adaptive patterns in different life situations.

At this point it is necessary to point out that we have never found evidence for impairment in verbal intelligence in long or short term alcoholics, although we have examined many groups (Tarter & Jones, 1971). On the other hand, we have found deficits in measures of visual-spatial and perceptual-motor abilities. These and numerous similar findings in the literature suggest a second neuropsychological alteration in chronic alcoholics i. e., that the right hemisphere

is more affected by chronic alcoholism than the left hemisphere.

Right Hemisphere Dysfunction

Our evidence for possible right hemisphere dysfunction comes from several sources. In a study which compared verbal and visual-spatial intelligence using the Shipley-Hartford and Raven's Progressive Matrices respectively we found the alcoholics compared to controls had significantly ($p < .01$) poorer Raven's scores but did not differ from the control group on verbal intelligence or abstracting ability (Jones and Parsons, 1972). Also in an unpublished study, we have constructed a verbal form of the WCST and found no deficits in the performance of alcoholics for control subjects. Finally, on the Raven's test when we subdivided the groups into long and short term alcoholism it is apparent (Figure 6) that the long term alcoholics are more impaired than short term. Interestingly the differences are greatest on items which are earlier in series. Other evidence of perceptual-motor deficits was found by Tarter (1971) when chronic alcoholics were given a visual-motor maze test and performed more poorly than control Ss but did not differ on a successive subtraction test.

In another study, (Parsons, Tarter & Edelberg, 1972) alcoholics had a significantly greater number of motor disturbances (tremor test) with the left hand than with the right hand ($t = 3.18$ $p < .001$). Within the alcoholic group the left hand was significantly poorer than the right while within the control group there was no significant difference. Finally, on the knob turning task described earlier, the largest differences between the alcoholics and controls were with the left hand ($p < .01$) while those with the right hand were in the correct direction but did not achieve significance.

Our evidence thus points to two likely sources of neuropsychological impairment in alcoholics especially those who have had 8 or more ye ars of alcoholism. The first is possible disruption of frontal limbic circuits and the second with possible right hemisphere dysfunction. As pointed out earlier both sets of deficits may be present outside of the awareness of the individual, yet profoundly affect his problem-solving and planning behavior.

In the presence of intact verbal intelligence, verbal concept formation and language communication skills, the alcoholic can present a picture of insight, understanding and communicative effectiveness. If the life tasks for the alcoholic were solely in these areas he undoubtedly would be effective. However, these strengths can give a rather unbalanced view of alcoholics' total behavior. When faced with conceptual problems of a non-verbal nature or certain types of perceptual-motor tasks the alcoholics can perform poorly, in fact, as poorly as persons who have known damage to the brain from other causes. The role of the frontal-limbic areas in the structuring of behavior over time, i.e. the plans and intentions for behavior has been described by Pribram (1972) Our data, especially that in which long term alcoholics interrupt successfully sequences of behavior, strongly suggest that in these patients, alterations in the frontal-limbic areas of the brain have occurred and the consequences pose problems for therapeutic or remedial endeavors.

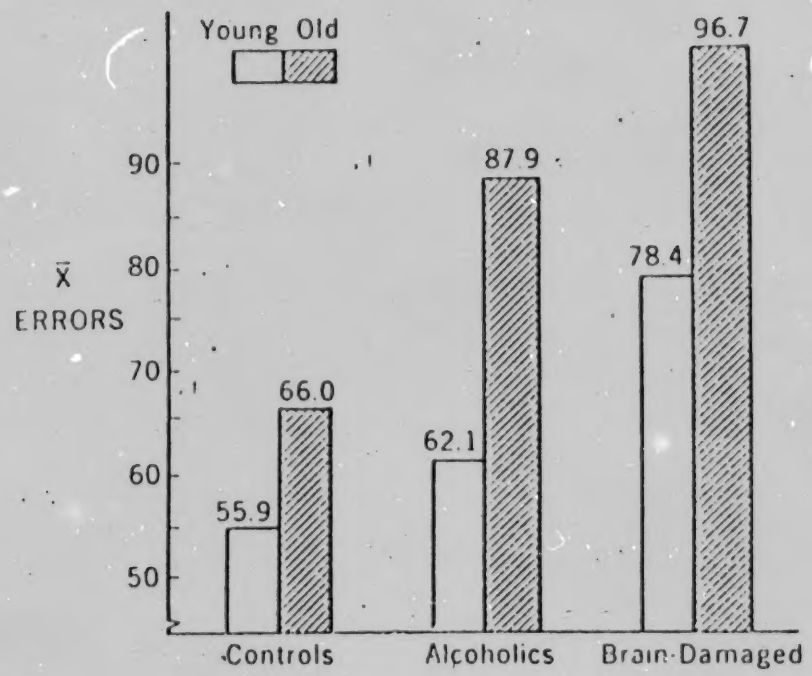

FIG. 1 - Halstead Category Test Performance

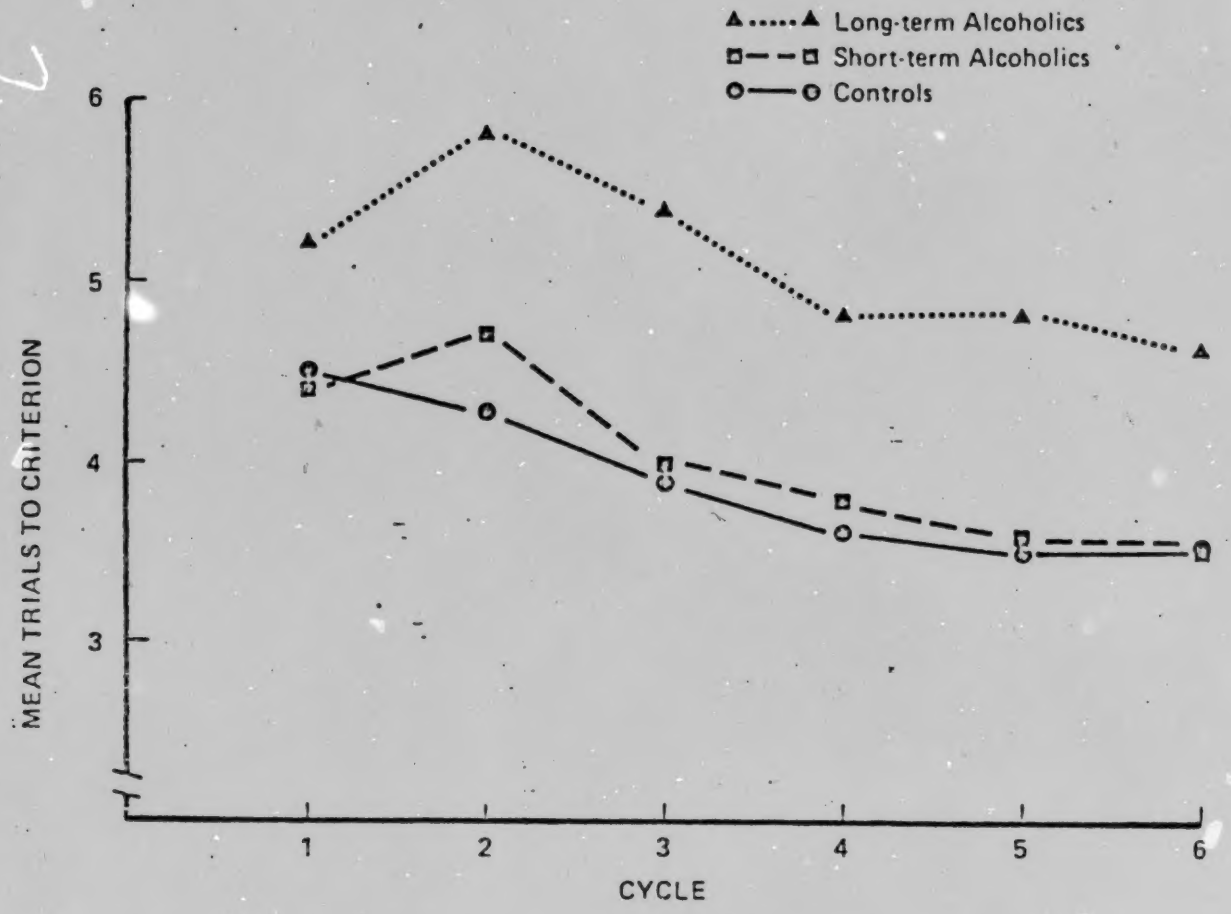

Figure 2. Mean trials to criterion on each cycle for the controls, short- and long-term alcoholics.

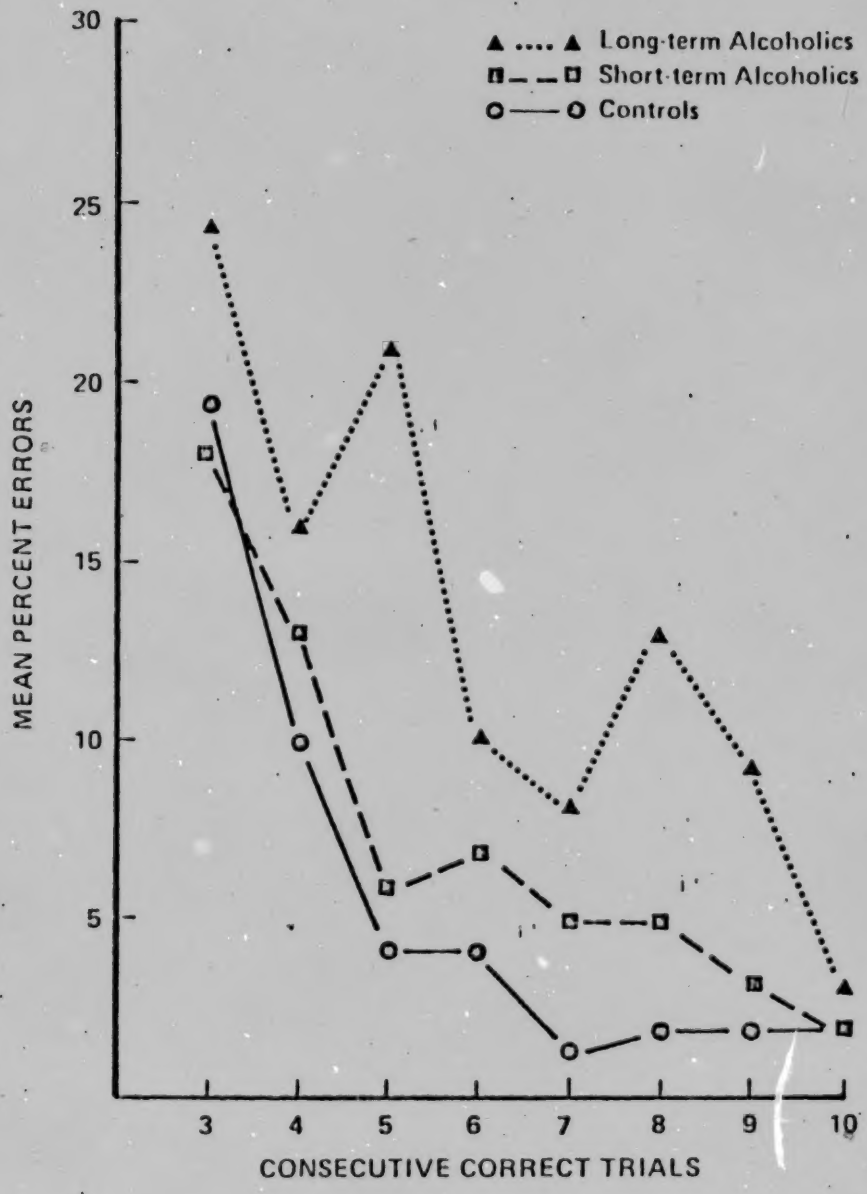

Figure 3. Percentage of times that an error succeeded a sequence of consecutive correct responses.

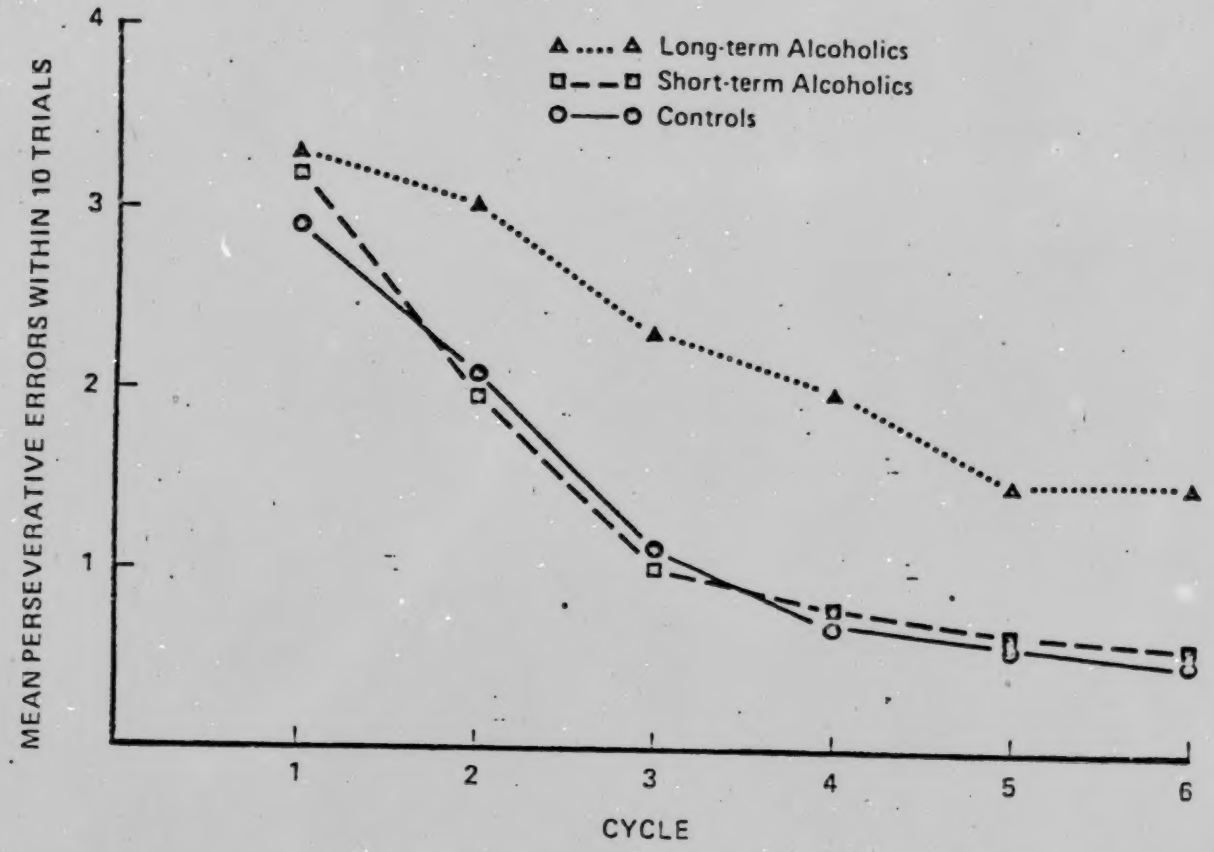

Figure 4. Mean perseverative errors emitted within the first ten trials after a set shift on each cycle for the controls, short- and long-term alcoholics.

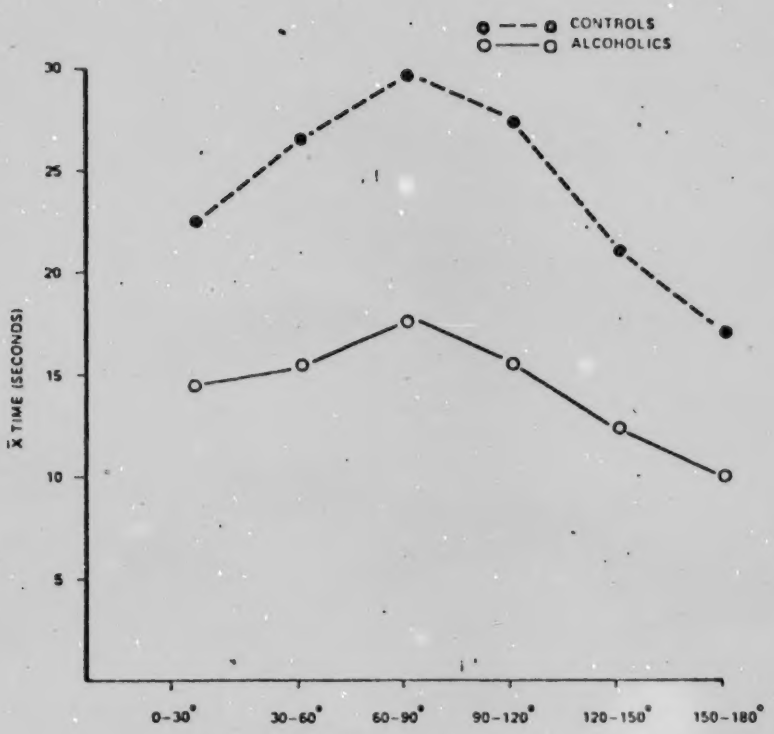
CONTROLS
ALCOHOLICS
30
25
20
15
10
5
X TIME (SECONDS)
0-30°
30-60°
60-90°
90-120°
120-150°
150-180°
SEGMENT OF ARC IN DEGREES

Fig. 5.

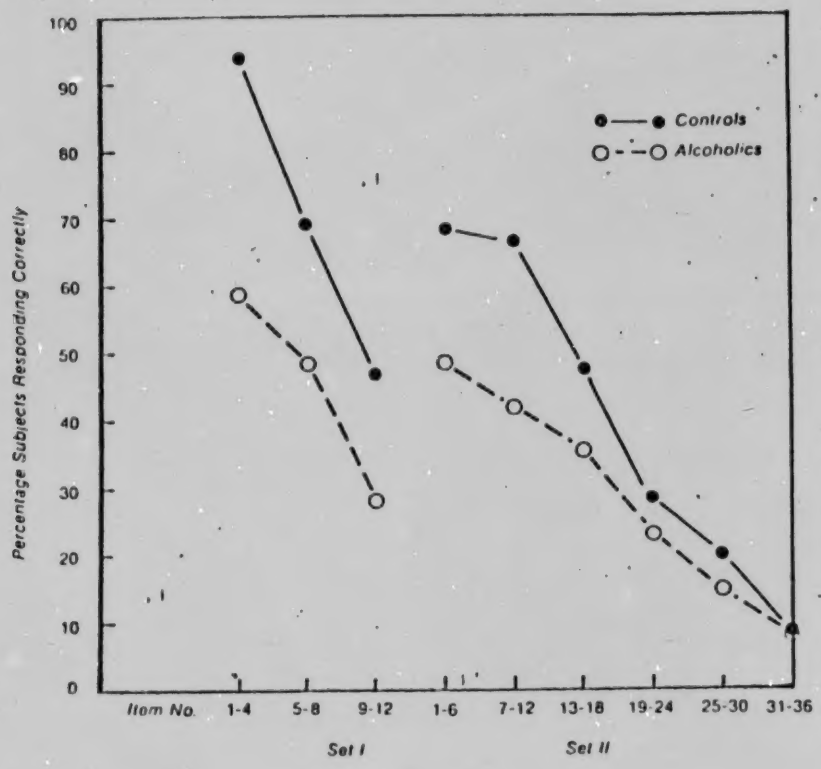

Fig. 6 Advanced Raven's Progressive Matrices (1962)

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FOOTNOTES

1. This paper was presented at the Southwestern Psychological Association Meeting, El Paso, Texas, May 3, 1974 as part of a symposium Altered States of Consciousness and Alcohol. The research reported was partially supported by USPHS, NIMH Grant 14702 and is the product of a group of researchers including Ben M. Jones, Ph.D., Ralph E. Tarter, Ph.D. and the author.

V. PHYSIOLOGICAL CONCOMITANTS OF THE ALCOHOL STATE: AROUSAL OR RELAXATION

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ABSTRACT: The present experiment was designed to discriminate among two diametrically opposed states, arousal and relaxation, which have been attributed to alcohol ingestion. Male social drinker subjects matched on age, drinking history, socioeconomic level and at least four hours food and stimulant deprived were assigned to form two independent groups of ten subjects each. Baseline measures of heart rate, skin conductance level, pulse wave amplitude and ear lobe temperature were recorded. Group I then received 0.3 ml/lb of body weight pure ethanol in 4:1 orange juice mix over a paced five minute period. Group II received an equivalent amount of orange juice per unit of body weight with 1 ml of ethanol floated on top. Physiological measures were continuously recorded over the next forty minutes. Groups did not differ on any of the measures during baseline suggesting that adequate matching on the stated parameters was obtained. Reliable effects were not obtained with the SCL measure. Heart rate increased reliably but nondifferentially for both groups. Ear lobe temperature increased for the alcohol and decreased for the placebo group. Initially, zero minutes post drink ingestion pulse wave amplitude decreased reliably and nondifferentially for both groups with respect to baseline. Five minutes post drink the alcohol group demonstrated a strong vasodilation effect which continued for thirty-five minutes. The placebo group stayed constricted and never reached baseline. It was concluded that in the dose used alcohol is a relaxant and that previous experiments found equivocal and conflicting results because they did not utilize a placebo control group, but drew inferences from within subject baseline to post drink comparisons.

Ludwig (1966, 1971) has comprehensively reviewed the techniques of inducing altered states of consciousness. He points out that drugs are one of the most reliable ways of manipulating state. James (1882) and Ritchie (1965) have observed that alcohol is one of the oldest and most often used drugs to manipulate state.

Psychophysiological techniques have been used for a number of years to index and evaluate a variety of states such as sleep stages, activiation and arousal levels, fear, anger and the physiological effects of alcohol ingestion (Naitoh, 1972). There is good agreement among experimenters that alcohol ingestion

induces an altered state as indexed by a variety of psychophysiological measures. However, there is less agreement among experimenters as to the exact nature of the relationship. Two diametrically opposed premises have guided much of the research relating psychophysiological measures to alcohol ingestion. The first premise holds that alcohol makes people feel relaxed and less tense (Carpenter, 1957; Greenberg & Carpenter, 1957; McGonnell & Beach, 1968; Kissen, Schenker & Schenker, 1959). It has been suggested that such alcohol induced tension reduction may be the motivation for the widespread use of alcohol. The second premise holds that alcohol ingestion leads to arousal and activation (Perman, 1958; Docter & Perkins, 1961; Morikawa, et.al., 1968; Walsh, 1971).

Earlier experiments have come up with equivocal and at times conflicting results, in part, because of methodological differences. Very few experiments have utilized placebo control groups, the same psychophysiological measures or the same dose of alcohol. Thus, both arousal and relaxation hypotheses are still being entertained. It was the purpose of the present experiment to discriminate between arousal and relaxation hypotheses of alcohol ingestion by utilizing a battery of physiological measures, a placebo control group and controlling for sex, age, drinking history, socioeconomic level and time since last ingestion of food and stimulants.

METHOD

Subjects. --The subjects were two independent groups of adult males matched on age, drinking history and socioeconomic status. Experimental and control groups did not differ reliably on any of the matching factors i.e., age 27.6 vs 26.0 years; income $6500 vs $5340; or alcohol consumption (the equivalent of three cans of beer or less/ week for both groups).

Procedure. --Subjects were told not to drink alcoholic beverages on the night before the experiments, to get a good night's sleep and to skip stimulants and breakfast.

Subjects came to the laboratory at least four hours food and drink deprived. After having the experiment explained to them subjects signed informed consent forms, were weighed and took a practice breathalyzer sample (to determine if in fact they had zero blood alcohol concentrations at the start of the experiment). Subjects filled out a questionnaire and were then instrumented to record heart rate, skin conductance, ear lobe temperature and photoplethysmographic pulse wave amplitude on a Beckman dynograph. Baseline measures were taken for ten minutes. Experimental subjects then received 0.3 ml of pure ethanol in a 4:1 orange juice mix/lb of body weight. Control subjects received an equivalent dose of orange juice per unit of body weight with 1 ml of ethanol floated on top of the drink. The drink was ingested over a paced five minute period. Autonomic measures were continuously recorded over the next forty minutes. Alcoholized and control subjects received a breathalyzer test at the end of the forty minutes to determine peak blood alcohol concentrations attained.

Response quantification

Skin conductance level. --Baseline SCL was measured as the mean of five once per minute samples at the 6th, 7th, 8th, 9th, and 10th minutes of the baseline period. Post drink SCL measures are based on the mean of nine measurements coming at the 0, 5th, 10th, 15th, 20th, 25th, 30th, and 40th minutes post drink.

Heart rate. --Baseline heart rate was quantified as the average of five ten second periods coming after the 6th, 7th, 8th, 9th, and 10th minutes of baseline. Post drink heart rate was quantified as the mean of nine ten second periods coming after 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink.

Heart rate variability. --Was defined as the range of heart rates in one minute periods during the 6th, 7th, 8th, 9th, and 10th minutes of baseline. Post drink heart rate variability was defined as the heart rate range in one minute segments 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink.

Ear lobe temperature. --Ear lobe temperature was taken throughout the experiment but was valid only in the drink and post drink period because prior to that Ss wore ear phones. Post drink ear lobe temperature was quantified as instantaneous readings 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink.

Photoplethysmographic pulse wave amplitude. --Baseline pulse wave amplitude (PWA) was recorded as the mean of five ten second samples 6, 7, 8, 9, and 10 minutes after start of baseline. Post drink PWA was quantified as the mean percent of baseline 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink (10 second samples).

RESULTS

Figure 1 summarizes the skin conductance results. A 2 X 2 factorial ANOVA was performed on the data. The between groups (experimental-placebo) factor was not significant. The trials (baseline-post drink) effect was marginally significant ($.05 < p < .10$). T Tests indicated that the placebo control group did not increase its SCL from baseline to post drink reliably. However, the experimental group showed a reliable baseline to post drink SCL increase ($t = 2.59$, $df = 9$, $p < .05$).

Figure 2 summarizes the heart rate results. A similar 2 X 2 factorial was performed on the heart rate data. The groups did not differ in average heart rate. Baseline to post drink heart rate increased reliably ($F = 47.75$, $df = 1,18$, $p < .001$). The groups x trials interaction was not reliable, suggesting that heart rate increased equally for both placebo and alcohol groups from baseline to post drink.

Heart rate variability did not differentiate between groups nor did it show any reliable changes over trials.

Figure 3 presents the photoplethysmographic pulse wave amplitude results as a percent of baseline. The results were analyzed by a 2 X 2 ANOVA. The groups effect was reliable ($F = 5.53$, $df = 1,18$, $p < .05$). The alcohol group was more

dilated than the placebo control group. The trials effect (zero and 40 minutes post drink ingestion) was highly reliable ($F = 25.90$, $df = 1, 18$, $p < .001$) indicating that dilation occurred overall. A significant groups by trials interaction was demonstrated ($F = 9.38$, $df = 1, 18$, $p < .01$). Figure 4 elucidates the nature of the relationship more clearly. Zero minutes post drink both alcohol and placebo groups showed a dramatic constriction such that response amplitude was only 50% of baseline. At this point the groups did not differ from each other. Five minutes post drink the alcohol group demonstrated a strong vasodilation effect while the placebo controls had not changed much at all. Thirty-five minutes later the alcohol group was still dilating while the control group showed a slight return toward baseline; a change probably due to relaxation in the experiment.

Ear lobe temperature data were analyzed by a 2 X 2 factorial analysis of variance. There was a significant groups effect ($F = 5.03$, $df = 1,18$, $p < .05$). No trial effect was demonstrated but the groups by trials interaction reached marginal levels of significance ($F = 3.85$, $df = 1,18$, $< .05$, $p < .10$). Further tests indicated that the groups did not differ reliably in ear lobe temperature zero minutes post drink ingestion. However, forty minutes post drink ingestion the alcohol group had a reliably higher ear lobe temperature than did the placebo control group ($t = 2.47$, $df = 1,8$, $p < .05$). The reliable difference forty minutes post ingestion came about because the alcohol group increased slightly and the control group decreased slightly, yielding in combination the reliable difference.

DISCUSSION

Results of the present experiment support earlier experiments in indicating that there are changes in autonomic activity symptomatic of an altered state after ingestion of alcohol when compared with baseline measures. The present experiment differs from prior experiments because of the addition of a placebo control group (subjects received the orange juice carrier medium plus 1 cc of ethanol floated on top). A baseline versus post drink comparison would

lead to the conclusion that ethanol increases tonic skin conductance levels. However, an experimental versus control group comparison does not support such an interpretation. Both groups recorded slight SCL increases which were neither differential nor reliable. Similarly, with the heart rate data, a comparison of baseline versus post drink heart rates would lead to the conclusion that alcohol has activating effects on heart rate. However, again such a conclusion is not supported by an alcohol versus placebo group comparison. Both alcohol and placebo groups showed reliable heart rate increases but there was no interaction to signify a differential effect. This finding suggests that the increased heart rates observed in both groups may have been due to cold orange juice in an empty stomach rather than a specific effect of alcohol. Such an interpretation of the heart rate data is in line with results reported by Docter and Perkins (1961) who also used a placebo control group and did not demonstrate differences between placebo and alcohol groups.

Since there were no significant group differences with heart rate and skin conductance measures, but pulse wave amplitude and temperature increased in the alcohol group while declining in the placebo control group, the results suggest that the specific effect of ethanol in the dose used is that of a relaxant. This is in line with verbal reports of the subjects suggesting feelings of warmth, drowsiness and relaxation. Further experiments are needed utilizing a dose-response analysis to test whether ethanol uniformly works as a relaxant or whether there are dose dependent arousing and or relaxing effects.

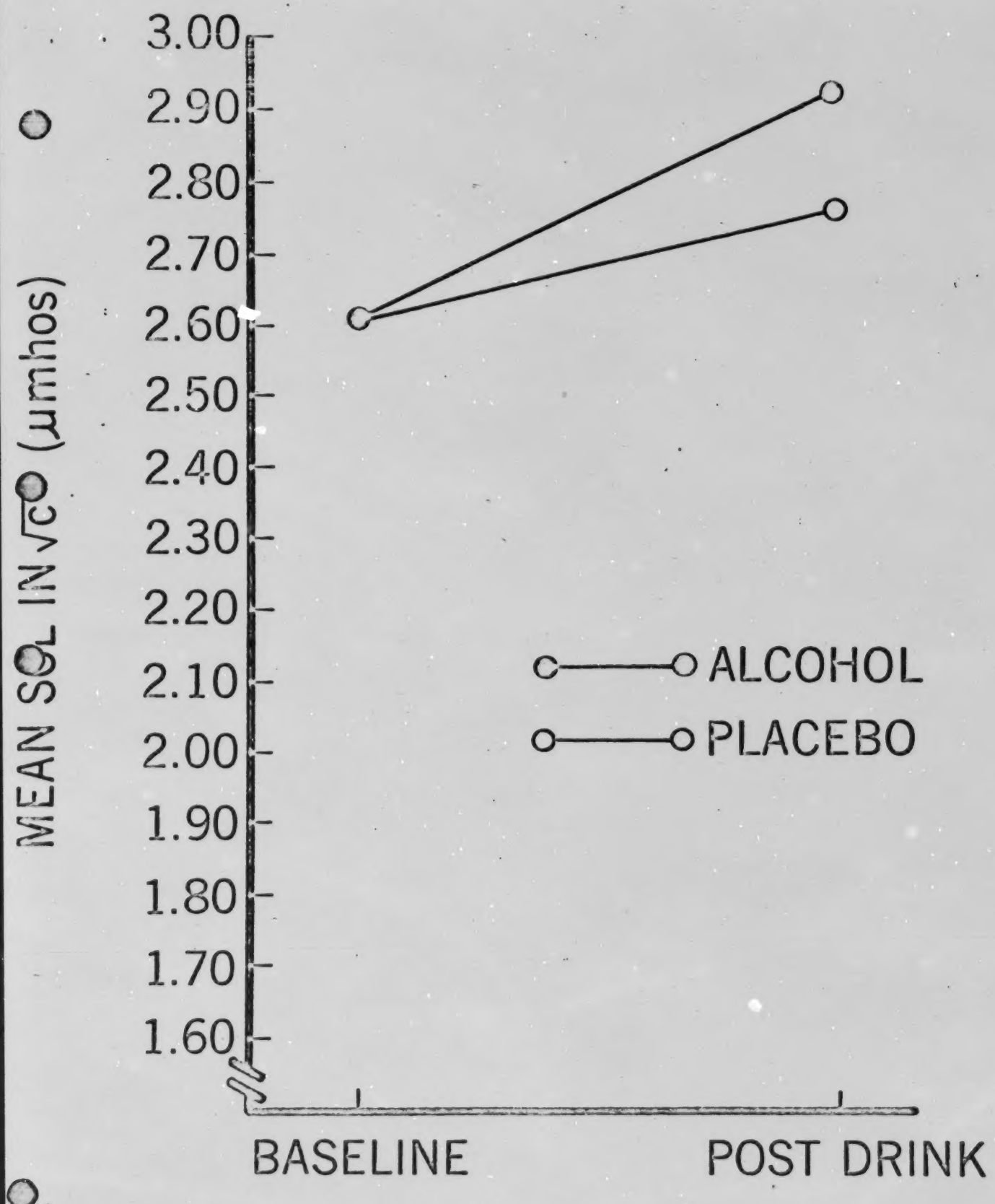

Figure 1. Mean skin conductance level at baseline and post drink conditions for alcohol and placebo groups (N = 10 each).

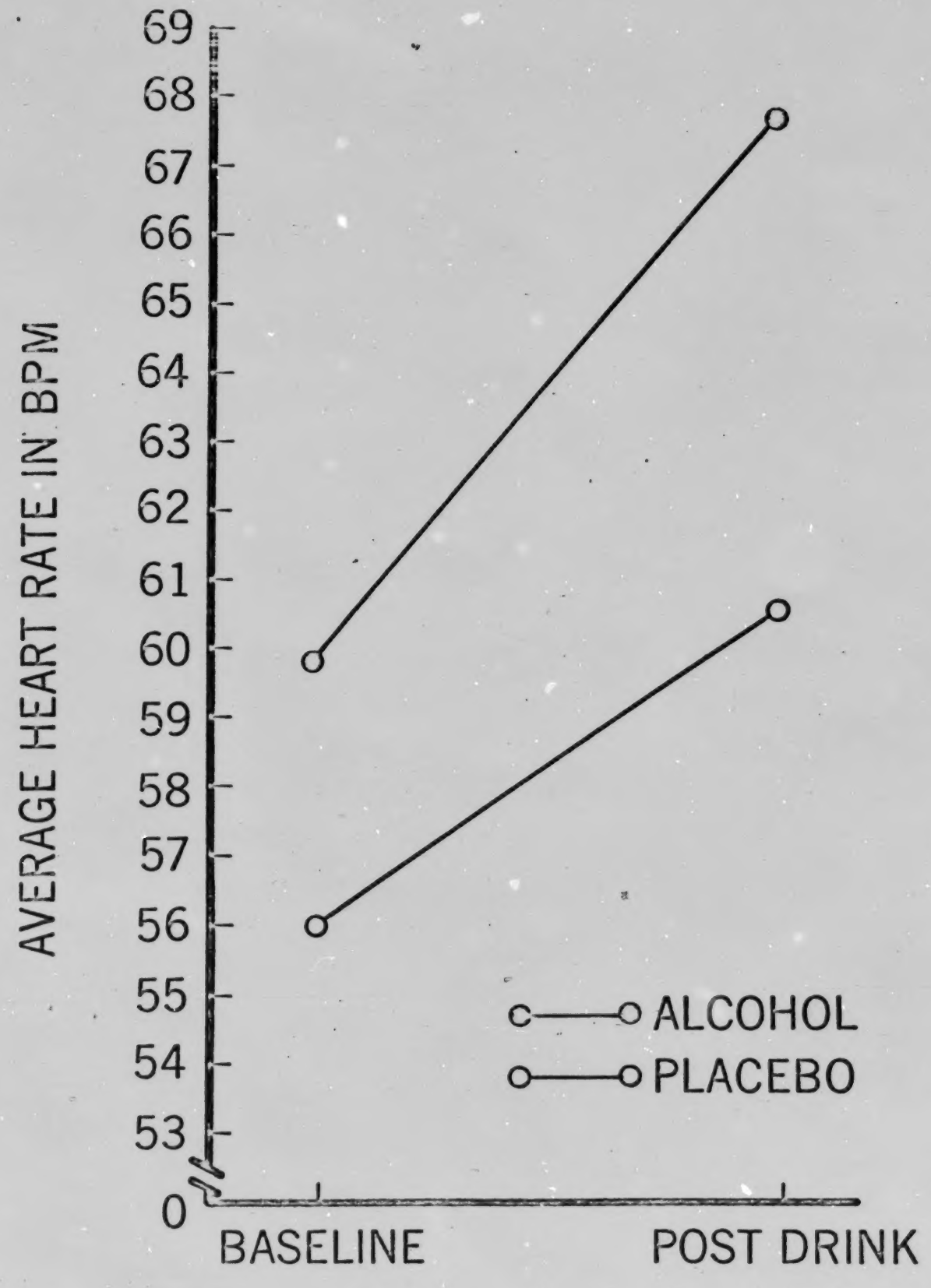

Figure 2. Mean heart rate levels over baseline and post drink conditions for alcohol and placebo groups (N = 10 each).

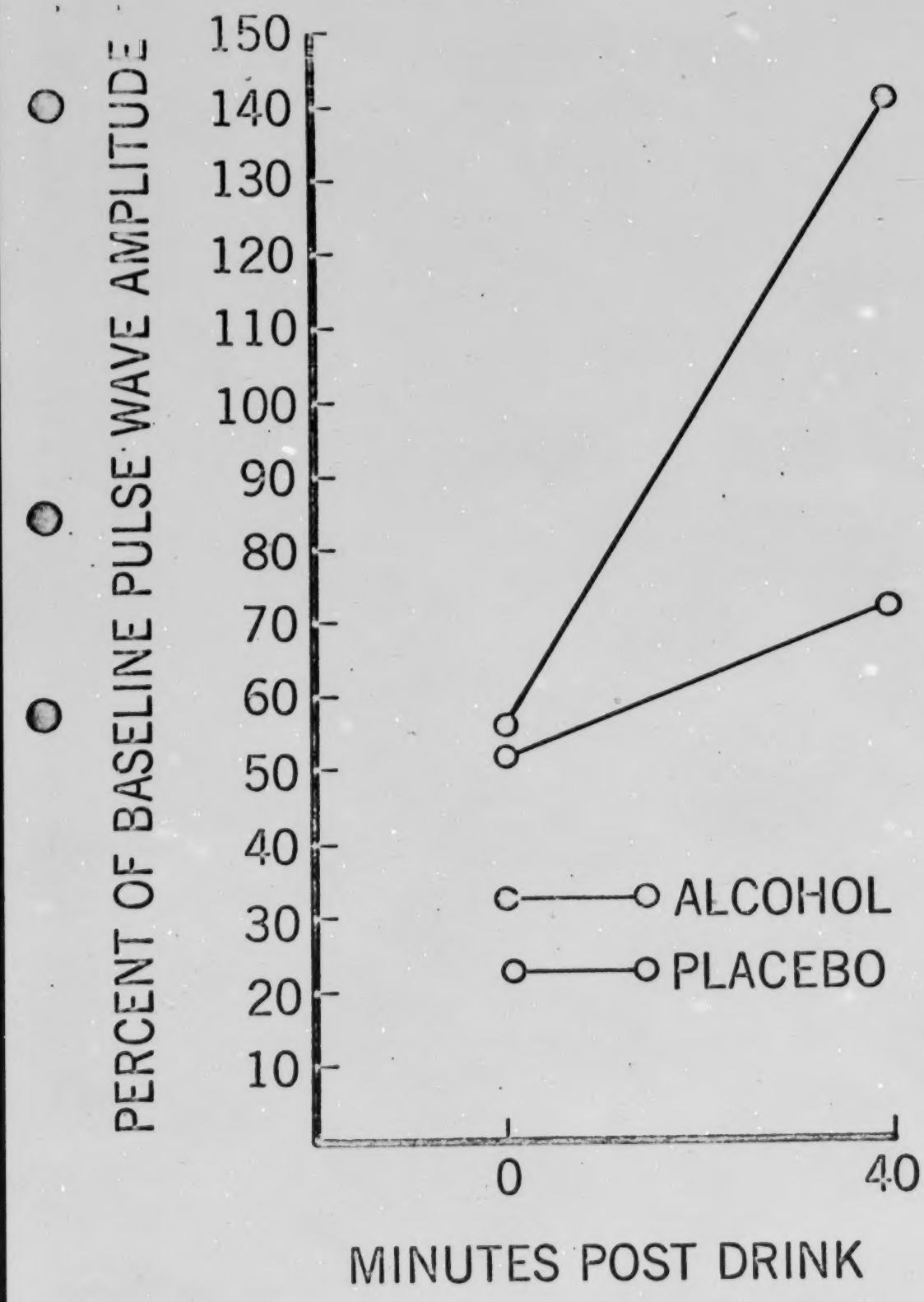

Figure 3. Percent of baseline pulse wave amplitude at 0 and 40 minutes post drink for alcohol and placebo groups (N = 10 each).

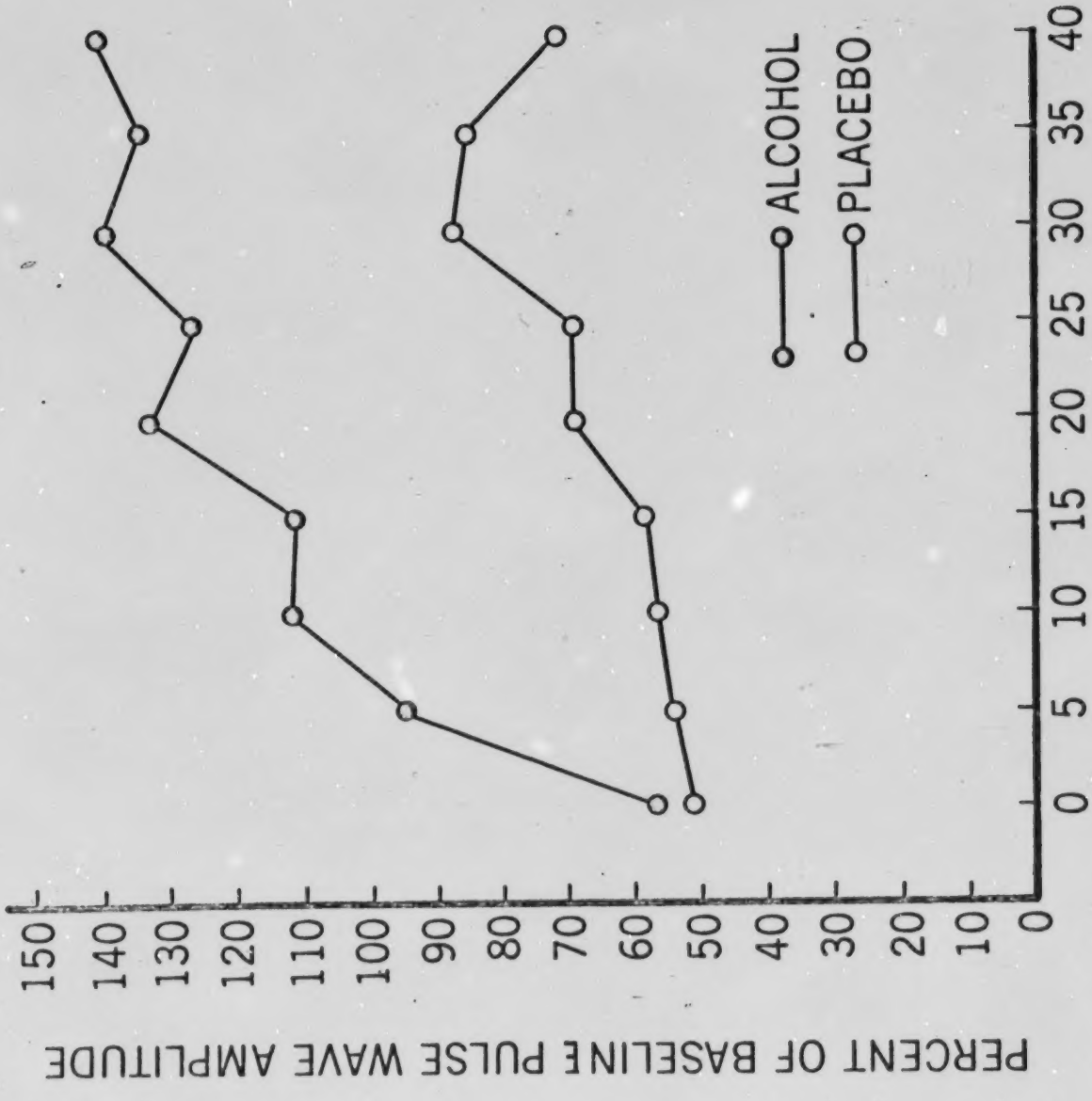

Figure 4. Percent of baseline pulse wave amplitude over five minute post drink blocks for alcohol and placebo groups (N = 10 each).

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VI. BIOLOGICAL RHYTHMS AND ALCOHOL EFFECTS:
ALTERED RHYTHMS OF CONSCIOUSNESS
A. Time of Day Studies and Menstrual Cycle Studies

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The purpose of this section is to report some of the factors that may influence how the individual will experience the effects of alcohol. There are many biological rhythms that vary throughout a 24-hour period and these are called circadian rhythms--for around a day. Body temperature is one such rhythm that has been related to performance throughout the day. Most individuals perform their best on cognitive tests during the afternoon when body temperature is usually high. It also has been reported that personality factors such as introversion and extraversion are related to both body temperature and test performance. Therefore, we decided to look at cognitive performance following alcohol (0.08% BAL) during the afternoon and evening to see if individuals reacted differently. Slide 1 (Fig. 1) illustrates that performance on the Raven's Progressive Matrices for a placebo group is slightly better in the afternoon than in the evening as would be expected. However, the opposite is found in two groups of subjects given alcohol. That is, performance was better in the evening than in the afternoon. This suggests that the state of consciousness produced by alcohol may be different depending on the biological rhythms at time of drinking. These same alcohol subjects demonstrated a significantly faster alcohol metabolism in the afteroon as compared with the evening as presented in Slide 2 (Fig. 2). Slide 3 (Fig. 3) presents the rate of alcohol metabolism for a group of chronic alcoholics during a drinking session in an experimental program. In this case, the high value during the afternoon reflects a slow alcohol metabolism, while the low value reflects a fast alcohol metabolism in the evening. This finding, opposite to that of the social drinkers, suggests that the rate of alcoholism may be altered throughout the day as a person becomes an alcoholic

and consequently drinks throughout much of the day and night. One might interpret these data to indicate the state of consciousness experienced by the social drinker and the alcoholic may be very much different since the interaction of alcohol with many biological rhythms may be altered.

Another biological rhythm, the menstrual cycle in women, also seems to interact with the effects of alcohol. Slide 4 (Fig. 4) presents peak blood alcohol levels for women tested at different times in the menstrual cycle. It is clear that most women reach a higher blood alcohol level and feel more intoxicated in the premenstrual period, about 3 to 4 days before they begin their period. These differences may be related to changing levels of female sex hormones, estrogen and progesterone, that take place at this time. These data suggest that alcohol may affect women differently throughout the menstrual cycle, producing what many women feel are unpredictable effects from alcohol. The unpredictability of the alcohol effects may protect women from becoming alcoholics as easily as men. Also, continued use of birth control pills, which maintain high sex hormonal levels, may lead to women becoming more vulnerable to alcoholism.

Two other factors may contribute to the effects or state of consciousness experienced from drinking alcohol. Slide 5 (Fig. 5) demonstrates that people who drink alcohol fast tend to absorb alcohol faster and eliminate it slower than those individuals who drink slowly. Slide 6 (Fig. 6) indicates that extraverts are more affected by alcohol than introverts on a cognitive task.

These data indicate that a variety of factors influence the way a person reacts to alcohol and the subsequent state of consciousness he experiences.

BIOLOGICAL RHYTHMS AND ALCOHOL EFFECTS:
ALTERED RHYTHMS OF CONSCIOUSNESS
B. Sleep Studies

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University of Oklahoma Health Sciences Center

For a number of years, we have been studying the effects of drugs (including alcohol) on various parameters of human sleep. The data that I am presenting today has to do with some of the physiological effects of acute alcohol ingestion and prolonged alcohol abuse.

One of the best documented aspects of sleep, both in humans and in subhuman species is that sleep is not a homogeneous, undifferentiated state of unconsciousness. Rather, sleep consists of a number of physiologically and behaviorally differentiable states ranging from drowsiness to the very deep slumber of stages 3 and 4 (sometimes called slow wave sleep).

In the 1950's, Dement and Kleitman noticed periodic rapid eye movements occurring during phases of sleep which electrophysiologically resembled light or Stage 1 sleep. Their subjects were difficult to awaken from this stage of sleep; and when awakened, the subjects usually reported dreaming. Thus, this stage of sleep became known as "dreaming sleep" or "Rapid Eye Movement" or "REM" sleep.

As stages of sleep came to be identified, a second important characteristic of sleep emerged: that is, the stages of sleep tend to occur in an ordered, cyclic fashion. Thus, when a person falls asleep, he will normally progress from stage 1 to 2 to 3 to 4 and then into stage REM. Usually this progression of stages will require about 90 to 100 minutes, and the cycle will be repeated several times throughout the night. The first cycle will consist of a good deal of slow wave sleep and a short REM period. As the night progresses, the amount of slow wave sleep in each cycle will diminish and the amount of REM sleep will increase so that by the end of the night, sleep will consist primarily of

alternations between REM and stage 2.

In general, acute doses of alcohol prior to bedtime have sedative effects, much like the barbiturates. Sleep onset is brisk, slow wave sleep is potentiated, and Stage 1 and Stage REM sleep is reduced. However, there is a considerable difference between the acute effects of intoxication and the effects of chronic abuse of alcohol. When dry and drug-free alcoholics were compared with matched controls, we found that the alcoholics had less slow wave sleep and more Stage 1 REM. (Table 1). In addition to differences in the relative amounts of the stages of sleep, the alcoholics had many more brief arousals, more frequent changes of stage, more disruptions of stage REM, and increased latency of sleep onset. (Table 2). In short, the sleep pattern of the alcoholic appears to be quite disturbed. The alcoholics seem to be experiencing what has been described as a "self-sustaining disregulation" of sleep.

The alcoholic can temporarily treat his sleep disturbance by drinking. When our alcoholic subjects drank before retiring, their sleep patterns, in terms of the distribution of stages looked very much like the controls. As shown in Table 3, intoxication also reduced the number of brief arousals and reduced the latency of sleep onset.

One way to assess the 90 to 100 minute ultradian rhythm is to compute the duration of the intervals separating periods of REM sleep. Previous studies in our laboratory has shown that, for young normal adults, moderate alcohol intoxication reduced the average inter-REM interval by about 10%.

The next slide (Fig. 1) illustrates the proporation of time intervals from REM offset to REM onset in a study of 20 alcoholics and matched controls. It is clear that the distributions are bimodal with one mode representing inter-REM intervals of 6 minutes or less, and a second mode appearing at 80-90 minutes. The modes to the left of 12 minutes represent the proportion of intervals which we classified as "REM disruptions" whereas the modes to the right represent the

basic period of the REM-to-REM cycle. The mode of the REM-to-REM cycle is about 15 minutes shorter in the alcoholics than in the controls, and the difference between the corresponding means is statistically significant. Thus, with regard to this particular ultradian rhythm, prolonged abuse of alcohol and acute intoxication produce similar effects; that is, an acceleration of the REM-to-REM cycle.

The next slide (Fig. 2) illustrates the effects of two nights of drinking in the same group of alcoholics. As with the young normal subjects, there was a tendency for the already foreshortened REM cycle to be further reduced, but this effect was not significant for the alcoholics.

There is still a lot that we don't know. The 90 to 100 minute ultradian cycle probably carries over into waking. However, we don't know much about waking biorhythms in either alcoholics or in intoxicated individuals. We don't know whether alcohol has similar effects on all biorhythms, or whether it produces biological desynchrony by accelerating some rhythms and not others.

One interesting note is that the sleep of psychiatrically depressed patients shows a markedly accelerated REM cycle; perhaps as short as 45 minutes. And the effects of at least one of the tricyclic antidepressants (desipraming) is to lengthen the REM cycle to about 90 minutes in these depressed patients.

Table 1

Stages of Sleep (percent)

		Controls	Alcoholics
Awake*	$\bar{X}$	10.7	10.1
	s	7.5	5.2
Stage 1	$\bar{X}$	5.4	10.5
	s	2.9	4.1
Stage 2	$\bar{X}$	61.9	58.1
	s	7.9	5.0
Stage 3	$\bar{X}$	9.4	4.2
	s	4.1	4.4
Stage 4	$\bar{X}$	2.8	1.1
	s	3.8	2.6
REM	$\bar{X}$	20.5	26.1
	s	4.3	3.2

* Means and standard deviations for awake are based on percent of total bedtime. All other entries are percents of total sleep without waking.

Table 2

Other Measures of Sleep

		Controls	Alcoholics
Brief Arousals (total)	$\bar{X}$	32.2	97.9
	s	24.9	27.5
Stage Changes (total)	$\bar{X}$	51.9	68.5
	s	13.3	14.0
REM Disruption*	$\bar{X}$	0.3	0.6
	s	0.1	0.1
REM Duration** (Min.)	$\bar{X}$	15.7	12.4
	s	4.7	3.4
Latency of Sleep Onset (Min.)	$\bar{X}$	20.9	31.7
	s	11.2	20.4

* Proportion REM offset to REM onset intervals of 12 minutes or less.

** Average duration of all REM episodes.

Table 3

Alcohol and Other Measures of Sleep in Alcoholics

		Baseline	Alcohol 1	Alcohol 2	Recovery
Brief Arousals (total)	$\bar{X}$	97.9	77.0	74.3	85.9
	s	27.5	23.6	24.9	44.8
Stage Changes (total,	$\bar{X}$	68.5	66.2	62.8	65.7
	s	14.0	16.9	13.6	29.1
REM Disruption*	$\bar{X}$	0.6.	0.5	0.5	0.5
	s	0.1	0.2	0.2	0.2
REM Duration** (Min.)	$\bar{X}$	12.4	11.4	10.9	13.7
	s	3.4	3.5	3.7	5.4
Latency of Sleep Onset (Min.)	$\bar{X}$	31.8	12.3	10.5	34.5
	s	20.4	11.6	10.6	22.2

---

* Proportion REM offset to REM onset intervals of 12 minutes or less.

** Average duration of all REM episodes.

FIG. 1

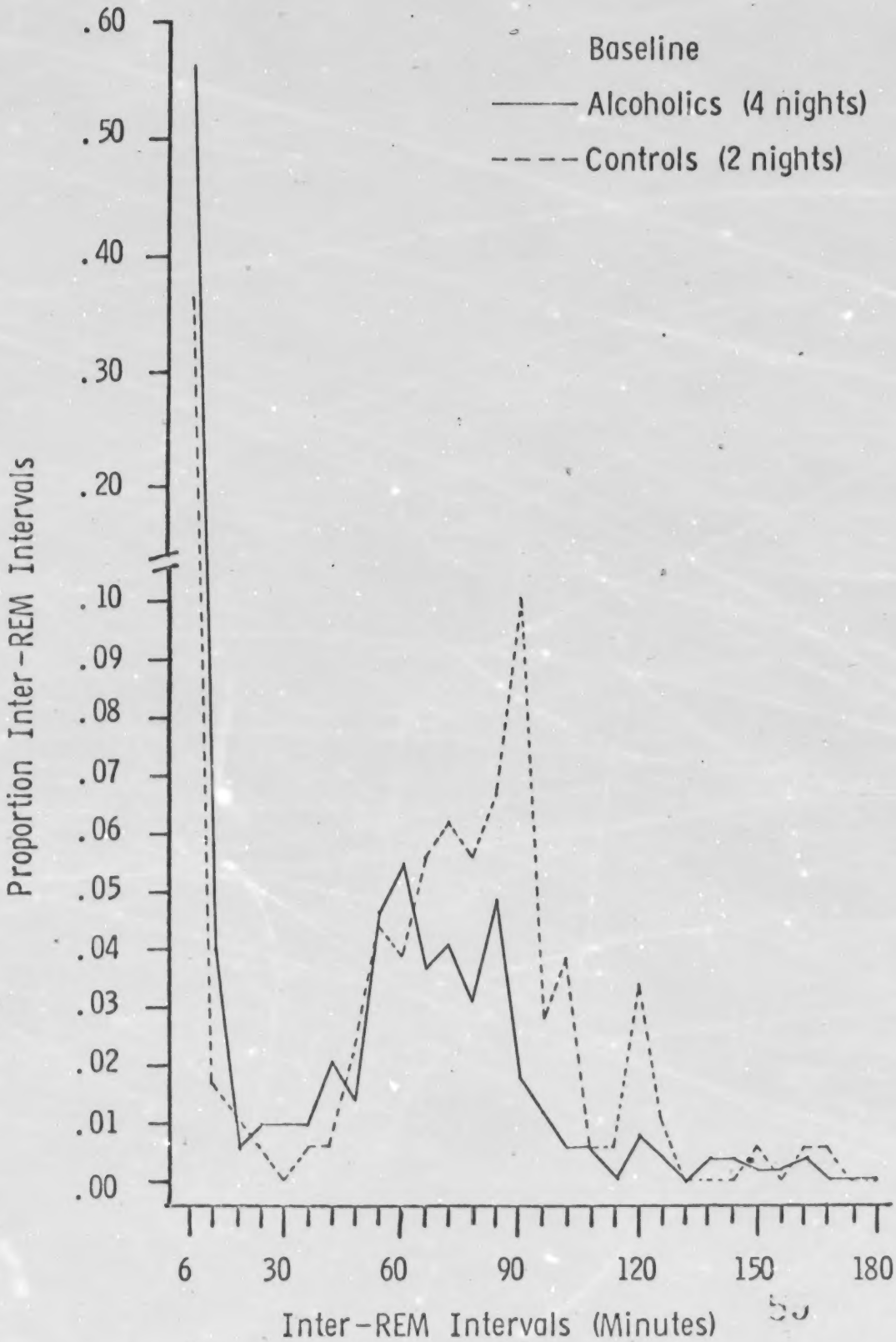
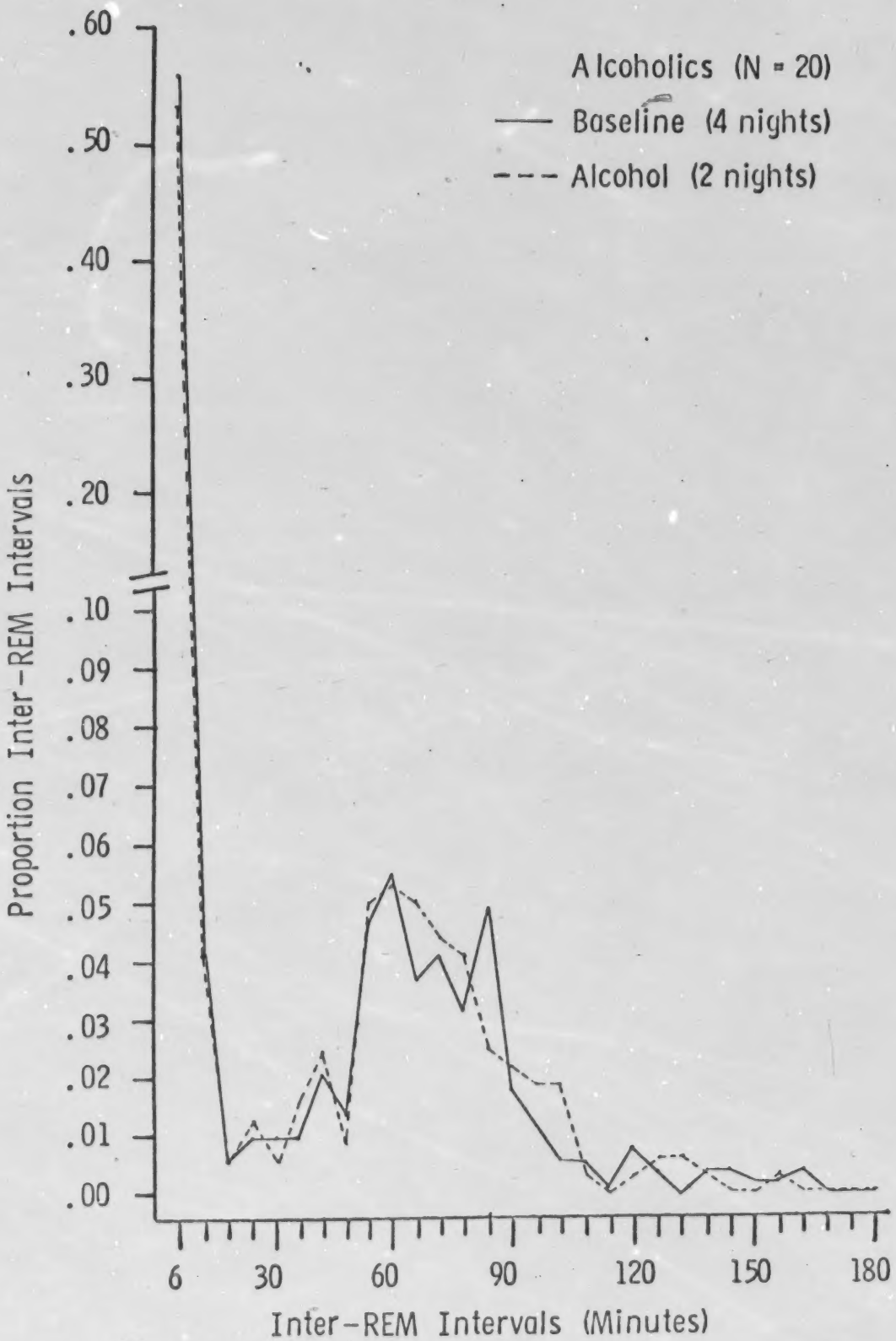

Baseline
Alcoholics (4 nights)
Controls (2 nights)
Proportion Inter-REM Intervals
.60
.50
.40
.30
.20
.10
.09
.08
.07
.06
.05
.04
.03
.02
.01
.00
6
30
60
90
120
150
180
Inter-REM Intervals (Minutes)

FIG. 2


Alcoholics (N = 20)
Baseline (4 nights)
Alcohol (2 nights)
Proportion Inter-REM Intervals
.60
.50
.40
.30
.20
.10
.09
.08
.07
.06
.05
.04
.03
.02
.01
.00
6
30
60
90
120
150
180
Inter-REM Intervals (Minutes)

VII. VOLUNTARY CONTROLS OF BLOOD ALCOHOL LEVELS: AN ATTEMPT TO MANIPULATE STATES OF CONSCIOUSNESS

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ABSTRACT: An experiment was conducted to document the ability of an alcoholic to discriminate levels of intoxication, to determine the rate of impairment of this discrimination and to measure the capacity to maintain prescribed levels of intoxication. In a series of nine weekly scheduled sessions, the subject received alcohol and was trained to discriminate his level of intoxication using a feedback technique. During periods of ad-lib drinking he was asked to maintain prescribed levels of intoxication for periods of one to two hours. The subject demonstrated that he could discriminate accurately his levels of intoxication. These estimates were equally accurate within 0.05% and 0.10% levels during the ascending limb of the blood curve. The discrimination was less accurate during the descending limb of the blood alcohol curve. Practice in estimation assisted by the feedback procedure improved the accuracy at all levels tested but improvement was greater at the 0.05% level on the descending limb of the blood alcohol curve. The alcoholic in our experiment was able to maintain prescribed levels of intoxication within two ranges, 0.05% and 0.10% for periods of one to two hours. Maintenance was more accurate at low levels (0.05%) but the greatest improvement was observed at 0.10% level.

The demonstrated ability to discriminate and maintain prescribed levels suggests that this alcoholic exerted considerable control over his drinking. It is possible that alcoholics drink to self-induce certain states of consciousness rather than compelled by an uncontrollable force.

Empirical studies suggest that, under appropriate conditions, alcoholics are capable to control their drinking behavior (Paredes, Cornelison, et al., 1968; Paredes, Ludwig et al., 1969; Nathan, Titler et al., 1970; Lovibond and Caddy, 1970; Cohen, Liebson et al., 1971; Mello and Mendelson, 1971; Gottheil, Corbett et al., 1972; Sobell and Sobell, 1973; Paredes, Hood et al., 1973). Control requires as an antecedent the ability to discriminate levels of intoxication. Given levels of intoxication could not be maintained if reference points were not available as a criteria to define them. Without a reference system deviation from criterion could not be detected. With this in mind we conducted an experiment to document this capacity for discrimination and to test the feasibility of devising exercises to improve the ability to maintain given blood alcohol concentrations.

The conceptual framework and empirical basis for this experiment is based on the work of Schachter(1968), Ekman, Frankenhaeuser et al. (1963), and Lovibond and Caddy (1970). Additional investigations with normal subjects (Bois and Vogel-Sprott, 1973) and with alcoholics (Silverstein, Taylor and Nathan, 1974) have generated data congruent with some of the assumptions implicit in our work.

The research paradigm was tested with a thirty three year old Caucasian male with well documented history of alcoholism. He had been hospitalized on numerous occasions and at various times he had exhibited symptoms of delirium tremens, blackouts and alcohol withdrawal. This person has a background of six months of college education. Occupationally, his last steady job had been as manager of the produce department of a grocery store. He had been arrested several times for public drunkenness and at the time of the experiment his driver's license was suspended for driving under the influence of alcohol. He lived in his parent's home where he assisted in the care of his ailing mother. The experiment was conducted on an outpatient basis. Permission to participate was obtained from the patient and his mother. The experimental sessions were scheduled once a week for nine consecutive weeks. To assure consistent cooperation the subject was transported from his home to the laboratory by the experimenters. The subject was instructed not to drink alcoholic beverages before reporting to the laboratory, a commitment that he kept in every instance. He had a light meal the night previous to each experimental day. Breakfast was withheld on the days of the testing. The subject usually arrived at the laboratory at 9:00 a.m. and was taken home at 5:30 p.m. His cooperation was excellent throughout and he seemed to look forward to each experimental session.

The objectives of the First Session were to familiarize the subject with a method of estimating blood alcohol levels following the administration of standard doses of alcohol and to obtain a baseline blood alcohol curve. These objectives were also explained to him; in addition he was asked to follow very closely any

cues or subjective feelings elicited by alcohol which could help him to discriminate his level of intoxication. These cues included physiological sensations such as changes in heart rate, respiration, feelings of warmth, tinglings, etc. The method used to teach him to estimate blood alcohol levels consisted of taking Breathalizer readings at five minute intervals. After each determination the subject was asked to estimate his blood alcohol level, following which the experimenter disclosed the actual Breathalizer value. This feedback information had the purpose of providing him with points of reference which he could use to improve the accuracy of his estimates. Appropriate precautions were taken to obtain valid Breathalizer measurements (Lovell, 1972).

At the beginning of the first session the subject received a dose of alcohol calculated to obtain a blood alcohol concentration (BAC) of 0.062%. Our previous experience indicated that this BAC is achieved by giving 0.66 ml ethanol/kg of weight. His weight was 67 kg. Therefore, he received 44.1 ml of 190 proof USP ethanol mixed with 176.4 ml of orange drink. This dose was to be consumed in five minutes. Two striking events occurred with the first dose of alcohol. A BAC peak level of 0.095% was reached which is much higher than we expected. Second, the subject did not appear to be much affected by this dose either subjectively or objectively. He experienced considerable difficulty in estimating his blood alcohol level because he did not seem to be able to detect the presence of alcohol in his system. After reaching the peak BAC, the concentration was permitted to decline to 0.055% at which point a second dose of ethanol similar to the first was administered and again the subject was permitted to drink it in five minutes. This manipulation had the purpose of determining if a higher ethanol level would facilitate blood alcohol estimates. A peak BAC of 0.16% was reached after which his BAC began to decline rapidly. As expected, the subject's estimates improved, becoming closer to the actual BAC readings as he approached 0.08% and continued to zero. Figure 1 illustrates the difficulties encountered in estimating blood alcohol levels

at the beginning of the experimental session and the surprising accuracy gained as the session progressed. These data suggest that this subject learned rapidly to make close estimates.

The Second and Third Sessions continued the training in blood alcohol level discrimination. A secondary objective was to determine if the subject could maintain blood alcohol levels within prescribed ranges for periods of two hours. In the first session the estimates were better at higher levels; therefore, a larger dose of ethanol, 1.32 ml/kg, was given at the outset of the Second Session. This amount was consumed within a prescribed period of fifteen minutes producing a peak BAC of 0.16% in fifty five minutes. At this point, the subject was asked to estimate his own blood alcohol levels using the same procedure as in the previous session. Two additional features were introduced to facilitate his estimates. A large board visually accessible to the patient was placed with the actual BAC readings plotted as well as his own estimates. This permitted a quick assessment of the degree to which his estimates coincided with the Breathalizer readings. Also a check list was completed by both the subject and the experimenter to evaluate the subject's mood changes, his physiological subjective changes and his subjective estimate of degree of intoxication. After the peak BAC was reached the level was permitted to descend to 0.11%. At this point a maintenance dose of the alcoholic beverage was made available to the subject. He was then instructed to maintain his blood alcohol level within a range of 0.13% to 0.16% for two hours and to consume only the amount needed to remain within the range. He also was informed that if the BAC reached a point beyond 0.16% drinking would be discontinued. The procedure allowed ten minute drinking periods followed by ten minute rest periods during which several breath samples were taken with the usual precautions. At the end of the two hours of the maintenance period, the subject was allowed to descend to zero. In this experiment, estimates of blood alcohol levels were obtained before, during, and after the maintenance drinking period. As Figure 2(a) illustrates, the

subject successfully maintained the blood alcohol levels prescribed by the experimenters. The same figure also indicates an improvement in the accuracy of the estimates of blood alcohol levels over the same performance during the first session.

Identical procedures were followed during the Third Session except that a smaller initial dose of alcohol was administered (0.66 ml/kg), and a lower range was selected for the maintenance period. The subject was allowed to peak at 0.08% and then descend to 0.06%, at which point he was instructed to stay within a range of 0.05% to 0.08%. The instructions indicated that if he exceeded 0.08% drinking would be terminated. On this occasion he was given five minute drinking periods followed by ten minute rest to allow for more breath samples. Figure 2(b) illustrates that the subject improved in his ability to estimate blood alcohol levels in spite of the fact that the performance was conducted at lower blood alcohol concentrations. Also documented was his capacity to maintain the prescribed level for two hours. His estimates during the descending limb of the blood alcohol curve are very accurate. In this experiment 132 ml of the alcoholic beverage remained at the end of the maintenance period. This indicates that the subject paced and discontinued his drinking in compliance with the instructions received.

During previous sessions the subject received Breathalizer feedback information every time his BAC was measured. The next logical step was to determine if the patient had developed internal reference points to make estimates. In the Fourth Session, to test this possibility, ad lib drinking with only limited assistance from Breathalizer readings was permitted. For the assessment a large amount of alcoholic beverage (the beverage contained 147 ml ethanol) was offered to the patient with instructions to drink only the amount necessary to reach 0.05%. Once he reported having reached this point, breath samples would be taken. He then would be asked to proceed to his estimates of 0.10% and then 0.15%. If his BAC

fell below the target level he could drink more to reach the expected level. On the other hand, if he went over the prescribed target value he could not continue drinking until he descended to it. If at any time he went over the target level by more than 0.03% drinking would be discontinued. On this session Breathalizer information was given only when the subject reported having reached the prescribed point and when the prescribed point and the actual Breathalizer reading coincided. Figure 2(c) demonstrates that the subject reported 0.04% when the actual Breathalizer reading was 0.03%. On the next drinking step he estimated 0.05% at an actual level of 0.06%. His BAC had decreased to 0.05% when he began to drink again to obtain 0.10%. This time he went over the target value reaching 0.12% and estimating 0.09%. He was allowed to descend to 0.09% and permitted to drink to obtain 0.15%. This time he estimated 0.15% when he actually was at 0.18%. At this point, the subject was permitted to decline to zero. In the session just described, the blood alcohol levels reached by the subject were only from 0.01% to 0.03% above the targets set for him. As in previous experiments his estimates were in general fairly accurate. It is important to note in this and the subsequent sessions the concentration of alcohol in the experimental beverage was varied from session to session to prevent the subject from relying on the volume of beverage consumed to make his estimates. Also a special pitcher and glass were used to prevent the subject from estimating visually the amount of beverage consumed.

In sessions Fifth to Ninth we combined the procedures employed previously by having the subject drink ad lib to reach predetermined blood alcohol concentrations and then request him to maintain prescribed levels for periods of an hour with Breathalizer feedback information available only at three points in the experiment. Throughout each of these experiments the subject had free access to the alcoholic beverage. He was asked first to

drink to reach 0.05%. When he reported to be at this point Breathalizer feedback information was given, and he was asked to maintain a 0.05% to 0.08% range for one hour without the assistance of feedback information. Then the subject was asked to drink to 0.10% level and to maintain 0.10% to 0.13% range for an hour also without feedback. This performance was thus programmed for the ascending limb of the blood alcohol curve. On the descending limb of the curve, the subject was asked to report when he had reached 0.05%, feedback was given and he was asked to maintain a 0.05% to 0.08% range, without having available any feedback information. Therefore, during the procedure he had only three anchorage points at which Breathalizer readings were presented: 1) when he reported that he was at 0.05% on the ascending limb; 2) after drinking to a reported 0.10% on the ascending limb and 3) when he had reported he had fallen to 0.05% on the descending limb. No other blood alcohol level feedback information was given during the experiment.

The data for these five sessions is illustrated in Figures 2(d) to 3 (a,b,c and d). It is apparent that the subject did very well at maintaining levels within the 0.05% and the 0.10% ranges on the ascending limb of the blood alcohol curve while he had some difficulty in estimating the levels when he was at 0.05% on the descending limb. Figure 4 presents the means and standard deviations of the BACs obtained during the maintenance periods and the blood alcohol level estimates reported at the beginning of each maintenance period. It may be observed that there was improvement over the five sessions in the 0.05% and 0.10% ascending limb maintenance periods with the subject maintaining a lower mean BAC and with a slight reduction in the standard deviations of the BAC. On the other hand estimates during the descending limb at 0.05% point were poor with the subject reporting 0.05% when he actually was at the 0.09% level. However, he maintained well this level even though it was the incorrect level. In order to help the subject improve on the descending limb he

was not allowed to begin drinking until he reached a BAC 0.05% regardless of the level he reported as being 0.05%. When the response was incorrect he was informed that the level was too high and he had to wait until he was at the 0.05% level at which point he was allowed to drink. An incident occured during session Eight. The subject estimated 0.09% as being 0.05%; he was asked to wait until he reached the actual 0.05% level but instead he drank to reach 0.11% and drinking was therefore terminated. On the following and last session the subject was asked to be more careful about the size of the sips he took. On that session, the subject performed very accurately. A possible source of deficient performance is that it requires a much smaller amount of alcohol to maintain the same level on the descending limb than on the ascending limb. Figure 5 illustrates the degree of accuracy with which the subject estimated his blood alcohol levels during maintenance periods. Difference scores were calculated between actual and estimated blood alcohol levels. A positive value indicates that the subject was underestimating the actual value. An improvement across sessions can be observed for the low and high maintenance ranges with the values gradually approaching zero. Although the same pattern of improvement is seen during the 0.05% descending limb maintenance period, there is marked underestimation except for the ninth session in which the subject made a very accurate estimate.

Tables 1 and 2 summarize the data obtained in the study. These data indicate that an alcoholic can be trained through an appropriate exercise to estimate and improve the accuracy of his estimates of blood alcohol levels, to drink to predetermined levels and to maintain prescribed blood alcohol levels for periods of one to two hours. The data also indicates that the alcoholic can exert control over his drinking behavior and modulate his blood-alcohol levels upon request.

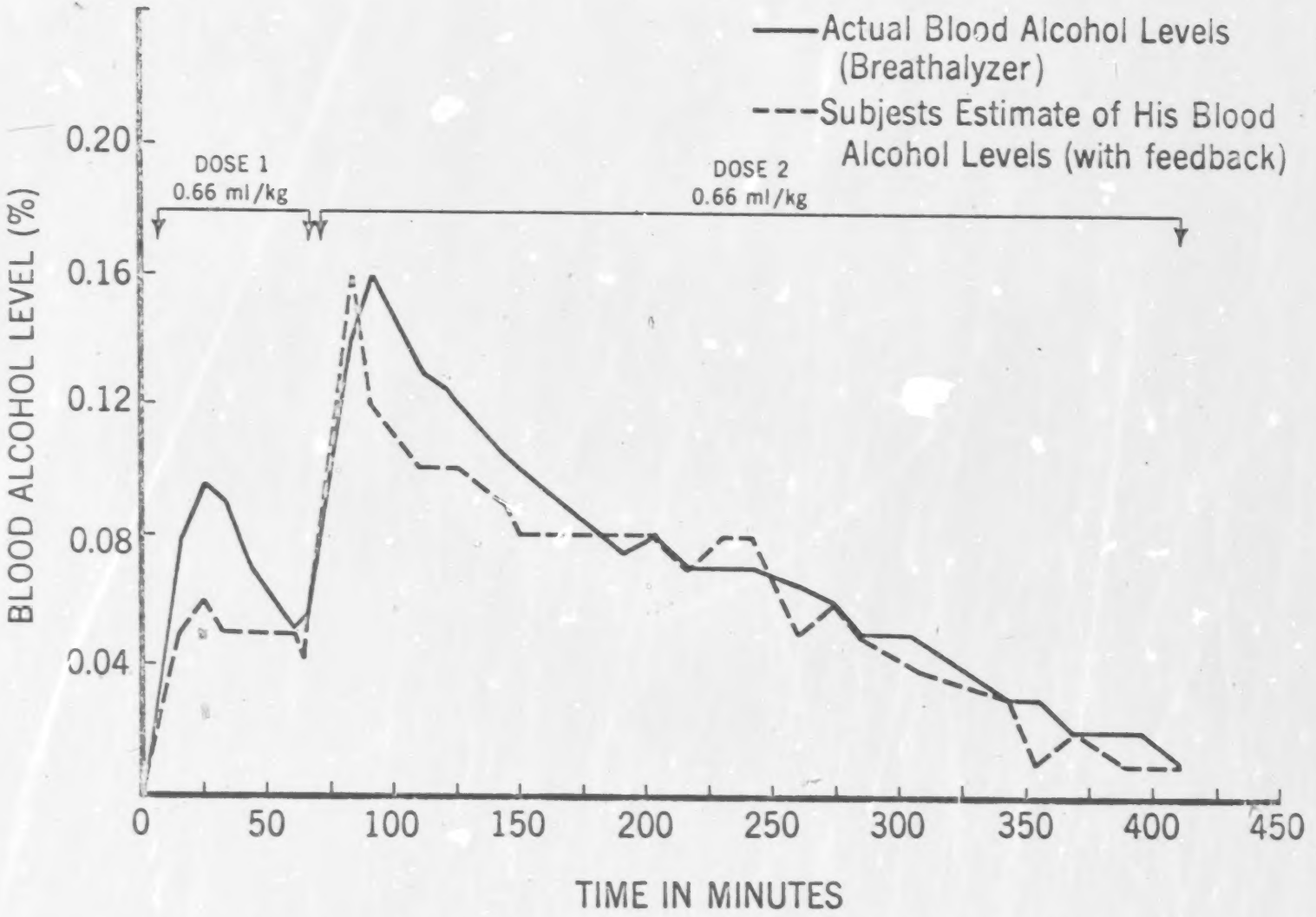

Figure 1. Blood Alcohol Data. First Experimental Session

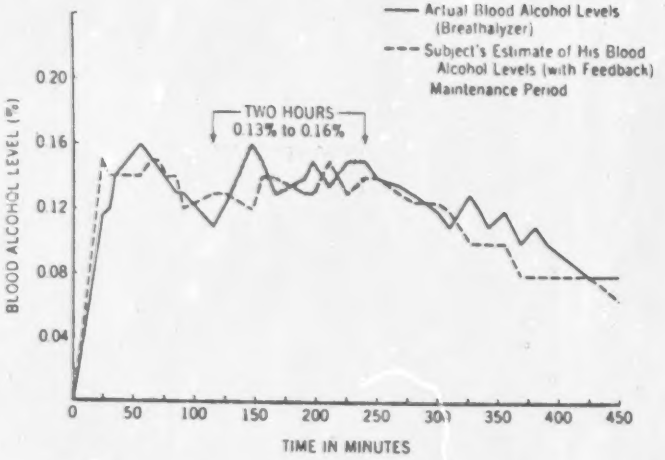

Figure 2 (a)

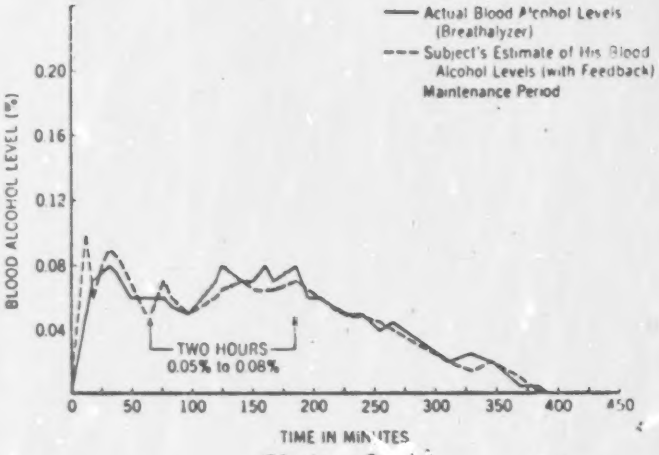

Figure 2 (b)

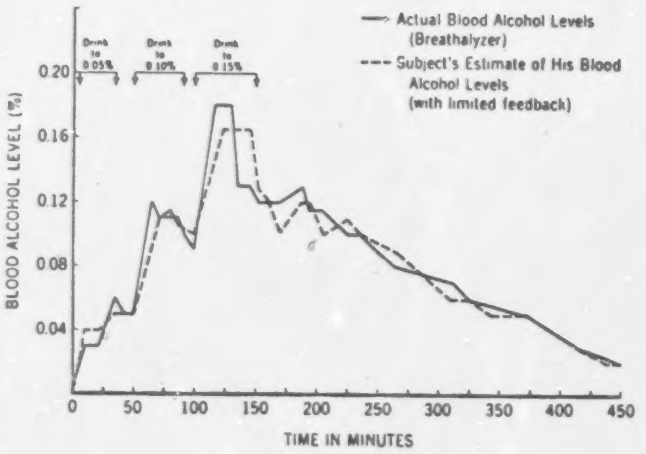

Figure 2 (c)

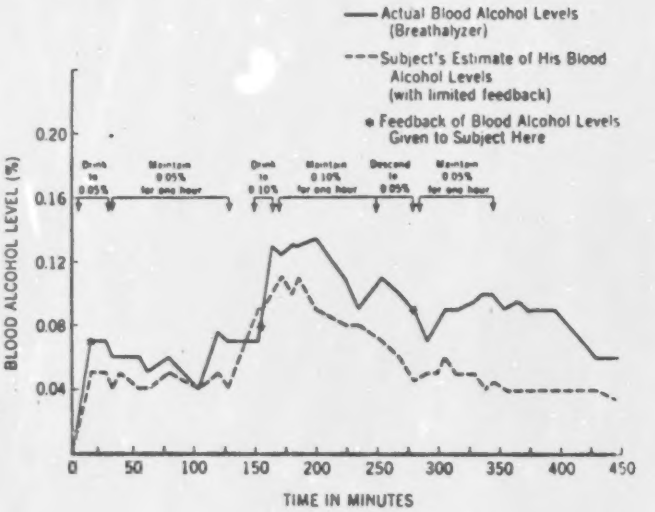

Figure 2 (d)

Figure 2. Blood Alcohol Data. Sessions Second to Fifth

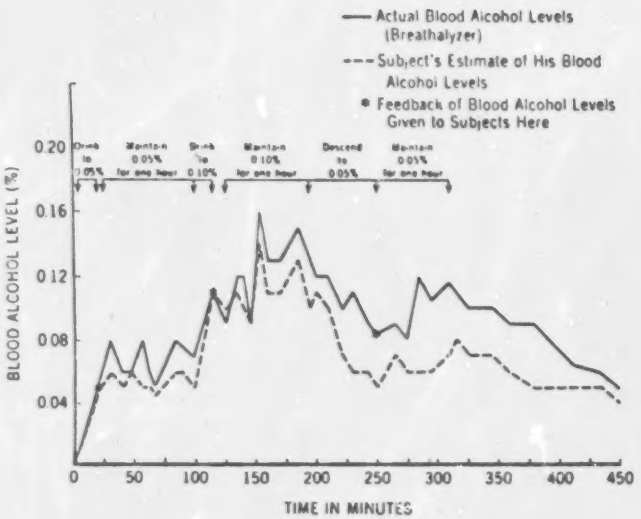

Figure 3 (a)

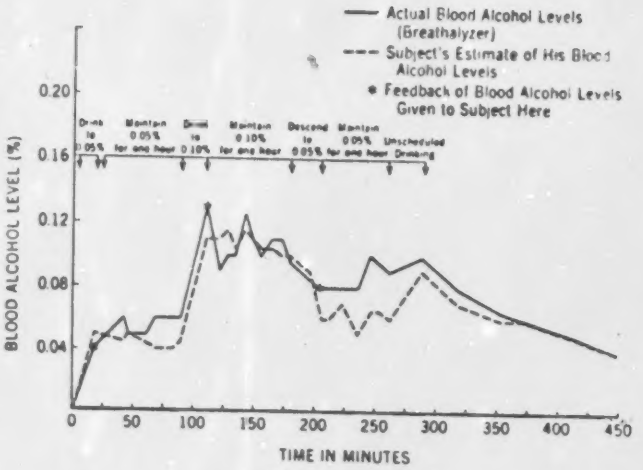

Figure 3 (b)

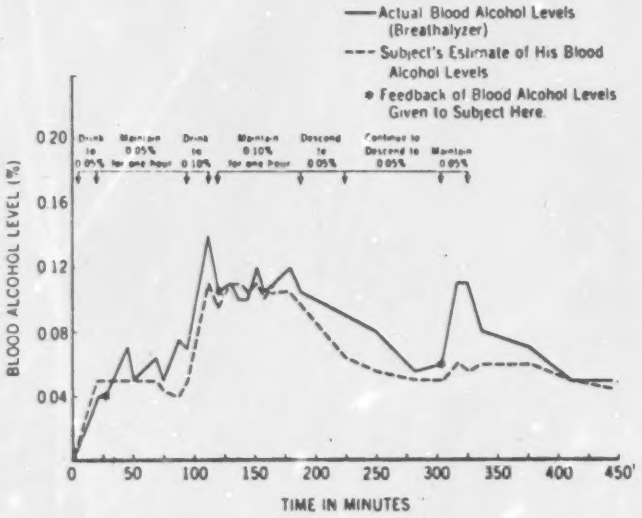

Figure 3 (c)

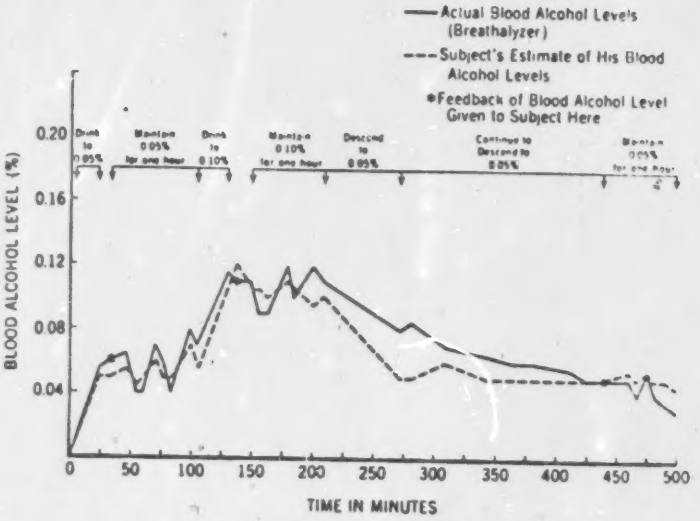
Actual Blood Alcohol Levels (Breathalyzer)
Subject's Estimate of His Blood Alcohol Levels
Feedback of Blood Alcohol Level Given to Subject Here
BLOOD ALCOHOL LEVEL (%)
0.20
0.16
0.12
0.08
0.04
0 50 100 150 200 250 300 350 400 450 500
TIME IN MINUTES

Figure 3 (d)

Figure 3. Blood Alcohol Data. Sessions Sixth to Ninth

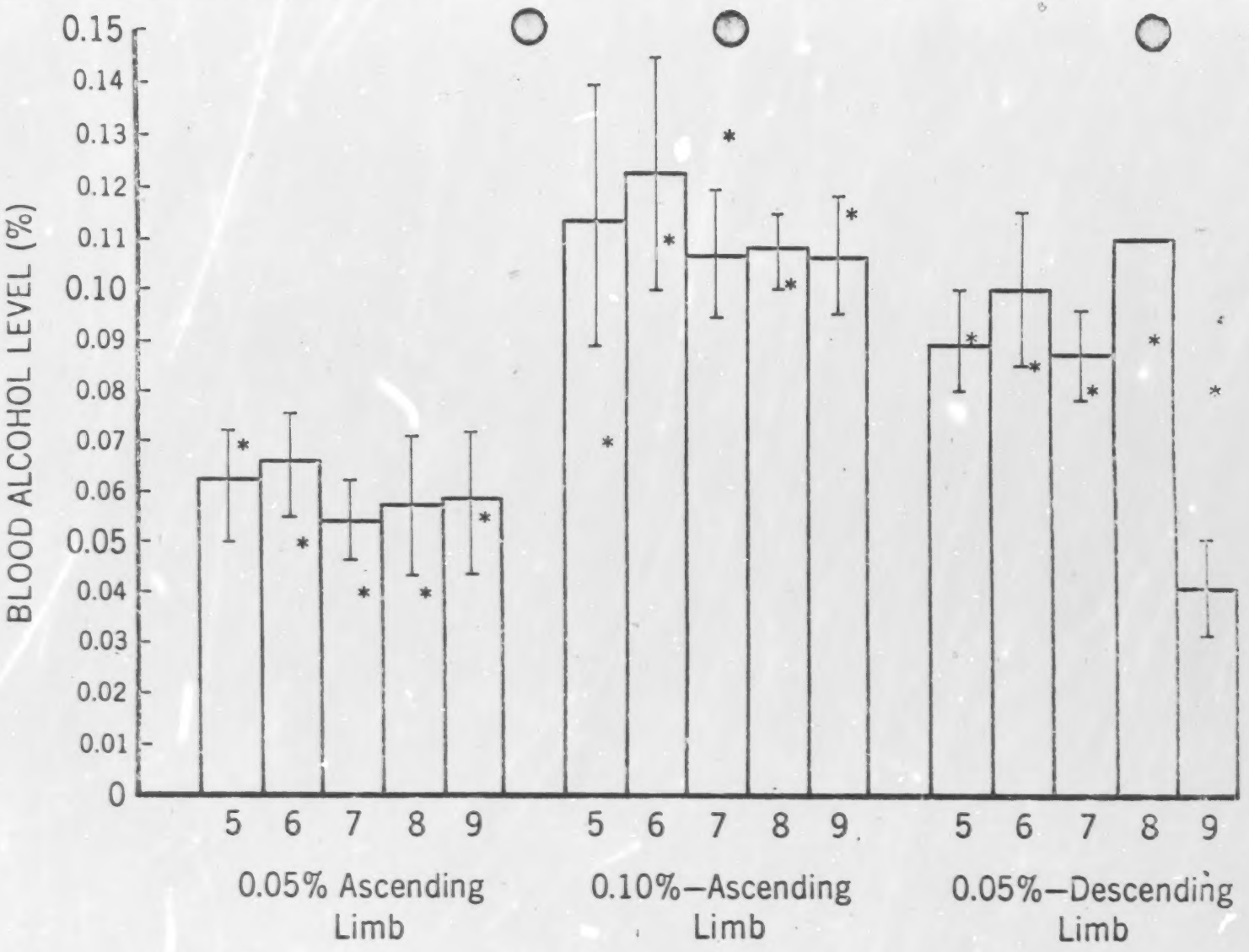

Figure 4. Means and Standard Deviations (Breathalizer) during maintenance Sessions Fifth to Ninth. Also, blood alcohol levels (*) reported at the beginning of each maintenance period.

Table 1

Blood Alcohol Data for Three Experimenter-Paced Drinking Sessions With Maintenance Periods

Session Number	Dose (ml/kg)	Ethanol (ml)	Drinking Time (Min.)	Peak BAC (%)	Time to Peak (Min.)	Two Hour Maintenance Periods			Totals For Drinking And Maintenance	
						BAC Range	Ethanol (ml)	Dose (ml/kg)	Ethanol Consumed (ml)	Dose (ml/kg)
1 (1)	0.66	44.1	5	0.095	24	-	-	-	88.2	1.32
(2)	0.66	44.1	5	0.160	90	-	-	-		
2	1.32	88.5	15	0.16	55	.13 to .16	44.25	0.66	132.75	1.98
3	0.66	44.7	5	0.08	32	.05 to .08	25.90	0.385	70.60	1.04

Table 2

Blood Alcohol Data for Five Subject-Paced Drinking Sessions With Maintenance Periods

Session Number	Drink Specific BAC (%)	BAC Obtained (%)	Time to BAC (Min.)	Ethanol (ml)	Dose (ml/kg)	One Hour Maintenance Periods			Totals For Drinking And Maintenance	
						BAC Range	Ethanol (ml)	Dose (ml/kg)	Ethanol Consumed (ml)	Dose (ml/kg)
4	0.05	0.06	35	-	-	-	-	-	106.6	1.59
	0.10	0.12	65	-	-	-	-	-		
	0.15	0.18	117	106.6	1.59	-	-	-		
5	0.05	0.07	16	-	-	.05 to .08	-	-	117.12	1.73
	0.10	0.08	155	-	-	.10 to .13	-	-		
	0.05	0.09	279	-	-	.05 to .08	-	-		
6	0.05	0.05	20	-	-	.05 to .08	-	-	124.80	1.82
	0.10	0.08	84	-	-	.10 to .13	-	-		
	0.05	0.085	250	-	-	.05 to .08	-	-		
7	0.05	0.04	18	26	0.40	.05 to .08	27.6	0.40	104.3	1.52
	0.10	0.13	111	30	0.44	.10 to .13	8.7	0.13		
	0.05	0.08	205	-	-	.05 to .08	12.0	0.18		
8	0.05	0.04	27	24.2	0.35	.05 to .08	29.23	0.42	105.0	1.52
	0.10	0.105	120	24.0	0.35	.10 to .13	15.20	0.22		
	0.05	0.09	224	-	-	.05 to .08	11.19	0.16		
9	0.05	0.055	24	30.5	0.44	.05 to .08	24.75	0.36	104.75	1.52
	0.10	0.115	131	25.25	0.37	.10 to .13	17.00	0.25		
	0.05	0.08	273	-	-	.05 to .08	2.25	0.03		

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VIII. DISCUSSION: ALTERED STATES OF CONSCIOUSNESS AND ALCOHOL

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The set of papers presented today bear on various questions related to alcohol and altered states of consciousness (ASCs). The paper by Tharp suggested that ASCs can be explored through an analysis of the stage or stages of information processing affected. The major finding was that alcohol seems to affect the response selection and organization stage rather than stimulus encoding or classification.

In the papers by Jones we learned of the influence of a number of variables on the effects of alcohol. For example, we learned that sex, phase of the menstrual cycle, time of day and limb of the blood alcohol curve all affect various aspects of behavior following alcohol ingestion.

Zeiner's work has emphasized the important methodological point that great care must be taken in attributing effects, in this case physiological effects, to alcohol without proper controls. The necessary control is one in which all aspects of the situation are the same for one group of Ss except for the actual ingestion of the drug. When this precaution is taken it appears that some effects attributed to alcohol, e.g. heart rate increases, occur in the group which received no drug, while other effects, e.g. ear lobe temperature and pulse wave amplitude variations, do seem to be caused by the drug.

The paper by Parsons on the effects of chronic use of alcohol suggested the interesting hypothesis that such use results in brain alterations which, however, do not affect the patient's state of consciousness. The defects produced appear to be in right hemisphere controlled functions (for example, visual-spatial or perceptual-motor performance) and in frontal-limbic controlled functions, i.e. those associated with the maintenance of set, and not within

left hemisphere mediated language areas which presumably are more closely associated with conscious awareness.

Finally, the paper of Paredes demonstrated a technique for dealing with the subjective identification of ASCs in a way which produces quantifiable data. The technique requires the S to establish and/or maintain various levels of intoxication when the only source of information available to S is his subjective awareness of the state.

With certain exceptions, in this group of papers one gets the feeling that certain underlying or covarying processes have been studied without the "it" itself coming directly under investigation. Within the tradition of experimental psychology this, of course, is not an unusual state of affairs. For example, we study learning, memory, motivation and perception frequently by testing constructs or physiological events presumed to underly the process and by observing quantifiable behaviors which presumably are reflections of the process. Not infrequently we define the processes themselves in terms of a set of measurable input-output relations in order to assure ourselves of the rigor and scientific character of the work. However, I think those of us who have engaged in this process will recognize that while something is gained, something is also lost in this approach to inquiry.

The shortcomings of this approach become especially obvious in connection with the study of ASCs because the topic, ASC, seems to demand a rather direct assault on the concept of consciousness itself. What is needed then is a willingness to deal more directly with consciousness which at this point seems likely to lead to exploration through personal experience or through the subjective reports of other persons who have experienced the states in question.

For this discussion let us consider that there are two approaches to studying consciousness and its alterations, one of which may be categorized as

scientific, predictive, quantitative and behavioral, and the other as scientific, descriptive, qualitative and experiential. Is one of these approaches better than the other? How do they relate to one another?

In order to deal with these questions I would like to pose another question which may clarify by analogy the original ones. Who has more knowledge about baseball, Henry Aaron, the player, or Howard Cosell, the sportswriter? My answer to this question is that they both have considerable knowledge concerning baseball, that they may have equivalent amounts of knowledge, but that the type of knowledge possessed by the two men differ. The sportswriter has knowledge _about_ baseball - who managed the Cubs in 1937, in what ballpark the most no-hitters have been thrown, et cetera. The player, on the other hand, has knowledge _of_ baseball - what it's like to run after a ball in the outfield, what it's like to hit a home run.

Getting back now to the two approaches to studying consciousness, I think the same kind of distinction is appropriate. The predictive, quantitative, behavioral approach gets at knowledge _about_ consciousness, and the descriptive, qualitative and experiential approach gets at knowledge _of_ consciousness. If I wanted to learn as much as I could about baseball I would check with both the player and the sportswriter and similarly if I wanted to learn as much as I could about alterations in consciousness, I would adopt both approaches - seeking knowledge _about_ and knowledge _of_ consciousness.

The concept of knowledge _of_ baseball or consciousness can be subdivided into direct and indirect knowledge of. If I listen to the subjective reports of the ballplayer or the alcohol user I am listening to verbalization transformations of their experience but not experiencing the experience. In this sense the knowledge _of_ is indirect. On the other hand, if I play baseball or drink alcohol I have direct "knowledge of" the experience or state of consciousness involved. Direct "knowledge of" is a private kind of knowledge, and there is

serious concern, notwithstanding the persuasive character of the knowledge, about whether and/or how this kind of knowledge can be incorporated into the body of science.

I would like to turn now to some comments about how combining the two approaches might lead to an overall gain in knowledge. Typical unimodal experiments, i.e. experiments without subjective reports, involve as <u>independent</u> variables, presenting alcohol to different classes of persons or patients, e.g. males and females, alcoholics and controls, under various conditions, e.g. various times of the day or at different stages of the menstrual cycle. These experiments might involve as <u>dependent</u> variables, performance on memory tasks, reaction times or physiological changes. Further, these experiments may implicate through theoretical analysis the stages of information processing affected or the type of memory deficit which occurs.

If these experiments also included assessment of subjective awareness of alterations in consciousness then a number of relationships could be explored. For example: What manipulations lead to particular alterations of consciousness? Are the alterations in consciousness the same for males and females? Are they the same for females at different stages of the menstrual cycle? Are either the quality of alteration or the occurrence of any type of alteration related to identifiable physiological changes? Is the stage of information processing affected by alcohol related to specific alterations in consciousness? Would other drugs or procedures which affect the same stages of information processing also produce the same alterations in consciousness? These and many other types of questions become answerable or at least posable if the two approaches are used in combination. There is also a more practical type of potential benefit to be derived from studying the subjective side of alcohol consumption. Our answer to the question, why does Johnny become an excessive drinker or an alcoholic, is frequently based on ideas concerning avoidance of something in the

environment. But avoidance of something always implies exposure to something else, in this case the altered state associated with consuming alcohol. Instead of focusing on what is avoided perhaps focusing on what is appealing about the altered state would be helpful in treatment. A clear understanding of these matters requires, I believe, a study of the subjective reports of these patients. One possible payoff is that what is attractive in the alcohol intoxicated state may be subject to change or it may be producable by alternative, less damaging means.

At this point I would like to briefly discuss some of the problems involved in the analysis of the subjective, experiential side of alcohol consumption. There appear to be two sets of problems. One of these is related to the use of subjective reports generally, and the other is related to the specific problem of subjective analysis of ASCs.

With regard to the first set of problems, scientific psychology has incorporated at various times in its history a variety of points of view regarding the acceptability of the subjective reports of experimenters or subjects. These points of view have ranged from acceptance of both kinds of subjective data as the cornerstone of scientific psychology to a rather complete rejection of such data in favor of quantifiable overt behaviors. One, perhaps more sophisticated analysis of the problem, has attempted to deal with the question of whether subjective reports should be considered, on the one hand, as a piece of behavior equivalent to any other behavior or as a special class of behavior endowed with meaning and more adequately reflecting the state of consciousness of the subject. Without further analysis of these questions I would merely like to point out that the pendulum seems to be swinging in the direction of acceptance of subjective reports.

In relation to the second set of problems, those associated with subjective analysis of ASCs, one pair of problems stands out because of the

implied impasse in which they seem to place the analysis. These problems are the state dependent character of various states of consciousness and the inability to verbalize the experiences. The state dependent problem implies that memory for the altered subjective phenomena would be unavailable in the "straight" state. This taken by itself does not necessarily impede analysis if subjective reports can be obtained during the altered state. However, the altered state may involve motivational changes which inhibit cooperation and furthermore, as suggested above, our language system, developed in the "straight" state, may not be adequate to enable the person to describe his experiences. The degree to which these problems in fact exist remains an open question. Even if they do exist it is not beyond the ingenuity of psychologists to develop techniques which get at ASCs and show that the alleged impasse is merely a temporary limitation.

The paper of Dr. Paredes provides a technique for getting at the detection and maintenance of ASCs in a direct way even though it does not get at the contents of the state. Perhaps, in order to get at the contents of ASC's we will have to develop new words to label the altered experience and learn to use them in a mutually agreed upon fashion. Whether this process will facilitate understanding or merely identification, or whether there is a difference between the two, is an exciting question which awaits further effort.